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# Temporal lobe epilepsy; a clinical investigation of psychopathology using computer-scored questionnaires

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Date

# Temporal Lobe Epilepsy:
# A Clinical Investigation
# of Psychopathology
# Using
# Computer-Scored Questionnaires

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# PREFACE

John Hughlings Jackson, in what was probably the first contribution of major significance to the study of temporal lobe epilepsy, wrote in 1888:

> He who is faithfully analyzing many different cases of epilepsy is doing far more than studying epilepsy. The highest centers ("organ of mind"), those concerned in such fits, represent all, literally all, parts of the body sensorily and motorily, in most complex ways, in most intricate combinations, etc. A careful study of many varieties of epileptic fits is one way of analyzing this kind of representation by the "organ of mind."(1)

A later neurologist, Frederick A. Gibbs, stated the problem more succinctly in 1951:

> To use a figure of speech, the sylvian fissure is one of the chief boundaries between neurology and psychiatry.(2)

I have tarried within this metaphorical fissure for the past twelve months, studying sixty-three cases of temporal lobe epilepsy, with the results that follow.

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1. Hughlings-Jackson, J. Brain 11:179 (1888)

2. Gibbs, F.A., J. Nerv. Ment. Dis. 113:522 (1951)

# INTRODUCTION

The problem presented is the probable relationship between the brain as an organ of the body and mental or emotional disturbance as a function of the organism. If we accept that the brain is the organ primarily involved in generation and regulation of mental and emotional events, albeit within the complex relations of the whole body, then disturbances in thought, mood and behavior can be studied through investigation of the brain and its functions. We have thereby reduced the problem to a logically consistent situation in which observation and recording of behavior, emotions and thoughts can be correlated with data on anatomy and physiology within a given person.

This introduces another problem, which I propose is largely a linguistic one. Correlation can only be undertaken within a coherent system of symbols and meanings, such as exist in the solution of algebraic or geometric relations. No such union of language exists in the solution of problems of brain function. One system has developed using the organism as the unit, and developed concepts such as id, regression and denial. Another system, using the cell or groups of cells as its unit, has developed such concepts

as action potential, synapse and spike. Each has its own techniques of measurement, valid for themselves but relating poorly to each other.

Although no one expects to localize the id within the brain or demonstrate personality disorder as a chemical deficiency, newer lines of investigation promise to generate a common language that will enable us to understand better the psychiatric problems that, to my mind, are the most uncomfortable, crippling and painful conditions to which the human organism is subject. Studies of biogenic amines and of sleep in depression, and of the role of hypothalamic structures in emotion and anxiety, are examples of correlations between neurological and psychiatric problems.

Another condition which has long fascinated neurologist and psychiatrist alike because of its position encompassing both disciplines is epilepsy, manifest as partial complex seizures with or without secondary generalization, arising in the temporal lobes and limbic structures of the brain. It is more commonly referred to as temporal lobe epilepsy, limbic epilepsy, or temporal-limbic epilepsy. These areas of the brain, as shall be discussed below, are implicated in memory, behavior, sensation, mood and motor activity. Clinicians involved with epileptics having this disorder have recognized a high incidence of psychiatric problems

as well as a tendency to poor control of seizures. In some patients the impression has been that seizures increase in frequency as the psychiatric problems become more severe. Above and beyond the life disturbance which epilepsy represents in itself, abnormal excitation of structures serving the functions mentioned above might reasonably have a role in the genesis of problems of behavior, mood and personality.

Thus a potentially fruitful approach to the problem originally stated, that of the relationship between neurological and psychiatric problems and concepts, would be the investigation of psychopathology in temporal lobe epilepsy and the delineation of the contribution of epilepsy itself to problems seen between seizures.

Hypotheses can be formulated bearing in mind the following assumptions:

1. Psychiatric disturbances are primarily mediated through brain structures.
2. Epilepsy and psychiatric disturbance, to whatever extent they are inherited, are not linked.
3. Drugs which act on the brain modify other functions besides those for which they are given.

At least three hypotheses are possible to account for psychiatric problems seen in temporal lobe epilepsy:

1. Sub-clinical discharging from epileptic neurons in the temporal lobe and limbic structures generate disorders of thought, mood or behavior by excitation of those areas of the brain mediating these functions.
2. The disruption of a person's life by frequent epileptic attacks, the social stigma of epilepsy, and the presence of chronic disease and disability alter a person's mood and self-concept sufficiently to distort personality, thought and behavior.
3. Sub-clinical epileptic discharges and problems of adaptation to life act through abnormally sensitive structures in temporal and limbic areas to generate together manifest distortions of personality, mood, thought and behavior.

If the first is a medical model and the second a social and psychological one, the third is an attempt to account for the many components of mental life and the several disciplines involved in its study.

The investigations reported in the body of this paper were conducted in order to generate data that would make possible an evaluation of the likelihood of these hypotheses.

## Chapter One

## TERMINOLOGY AND PRESENTATION

### A. History

Hippocrates, the "father of medicine," is sometimes credited with the first description of temporal lobe epilepsy. Lennox quotes him as follows:

> And I see men become mad and demented from no manifest cause, and at the same time doing many things out of place;---and this will happen not once but frequently. (1)

Lennox further describes hallucinations of sight, smell and hearing "near the accession of the paroxysm" from the writings of Aretseus in the second century, and a report by John of Gaddesden in 1314 of an automatism in which the patient would hold his forehead, rub his face, stand up and recite the Lord's Prayer, and finally spit once, which "threw off the paroxysm."

There are many reports from the nineteenth century of epileptic mania, epileptic delirium, "psycholepsy," and "psychical equivalent," described by Lennox (1). It was generally accepted that epileptics might show these symptoms of unusual sensation, consciousness or behavior, but

it was not clear whether these were "epileptic fits," or whether they could be the sole manifestation of epilepsy. Reviewing his own cases and cases from the literature, Hughlings Jackson in 1888 addressed himself to these questions.

Jackson divided the observed behavior into motor aspects, the automatism, and the "intellectual aura," which he renamed the "dreamy state." He pointed out that the dreamy state was both a defect in consciousness and an over-consciousness, whereas automatism was generally accompanied by loss of consciousness. Contrary to some of his colleagues, he considered the dreamy state to be a "slight epileptic fit" and not an aura, thereby different from "crude sensations ('warnings') of (a) smell or (b) taste." The dreamy state would occur sometimes with these crude sensations or with an automatism or both, but might occur by itself. Jackson was very reluctant to diagnose epilepsy solely on the basis of the dreamy state because of its similarity to "reminiscence" seen in non-epileptic people, generally requiring to see convulsions in addition. However, paroxysmal occurrence of this dreamy state, especially when very frequent, would have made him suspicious and he would have begun treatment if consulted. (2)

In the following years several reports of anatomical involvement of the temporal lobes with tumor, atrophy, scarring and softening were published, reviewed by Liddell (3). Gowers in 1881, and also Crichton-Browne in 1895, described the sensation of fear or horror, different from a natural fear of an impending seizure, which sometimes accompanied the dreamy state. Wilson in 1928 classified the dreamy state into four groups, familiarity (deja vu), unfamiliarity, panoramic memory and incomplete types. He suggested that they may merge into one another and when combined with hallucinations of taste or smell comprise an "uncinate fit." He further considered these symptoms to be an aura and not a fit in themselves. (3)

Three studies published in the 1930's proved to stimulate new ideas and results along three separate lines. The hypothesis that seizures and schizophrenia are opposites, proposed by Meduna and reviewed by Flor-Henry recently (4), led to the induction of seizures as a form of therapy. The fact that this treatment was least successful in patients least responsive to other forms of therapy suggested that induced convulsions delineated a population with good prognosis and a tendency to convulsive phenomena. More importantly, as will be discussed below, it became apparent that certain forms of epilepsy predisposed to

psychoses. Thus the hypothesis (but not the therapy) fell into disfavor, although recently restated by Reynolds (5).

The introduction of the electroencephalogram allowed a clinical-physiological correlation to be attempted, as was done by Gibbs, Gibbs and Lennox in 1938. Gastaut reviewed their results in 1953, pointing out that the initial correlation was between episodes of clouding of consciousness or amnesia with automatism or emotional disturbance, and an EEG abnormality of generalized six cycles per second slow wave activity, the waves having wide amplitude and a flat top (6). The conception that this entity, which they named "psychomotor" epilepsy, derived from deep midbrain structures was supported by this generalized abnormality in the EEG. The Gibbs' refined their technique, reported both "fast spikes" and "slow spikes" of positive deflection in psychomotor epilepsy, but continued to record unipolar electrodes against the ears as reference. In 1948 they reported different findings when the ipsilateral electrode was disconnected:

> The generalized positive spikes disappear, or are greatly decreased in amplitude, and the focus of negative spikes stands out clearly in the anterior temporal area. (7)

With this technique or the addition of bipolar recording or nasopharyngeal leads, Gibbs, Gibbs and Fuster were

able to assign a temporal lobe origin to the focus in all of 300 cases of "psychomotor seizure discharge."

The third line of investigation was stimulated by the publication in 1937 of Papez' theory of the limbic structures as elaborating emotions (8), and by the results of Kluver and Bucy in 1939 (9) on experimental ablation of temporal lobes and limbic structures in monkeys. A very productive series of publications ensued, generating a physiological concept of the function of these areas, and is reviewed below.

Thus by the early 1950's it was possible to speak of "temporal lobe epilepsy" having those aspects recognized by Jackson: various psychomotor phenomena, automatism with amnesia, and subjective experiences including the dreamy state and sensory hallucinations (10). Lennox, referring to the same set of characteristics, delineated a "psychomotor triad", but suggested the greater accuracy of the term "temporal epilepsy." (1) In a massive critical study published in 1953, Gastaut reviewed the problem of "psychomotor" and "temporal" epilepsy in the light of his own experimental findings (6). He recommended subdividing the disorder into three varieties: temporal psychomotor epilepsy arising in the temporal cortex, hippocampal psychomotor epilepsy arising in the hippocampus or adjacent

areas, and diencephalic psychomotor epilepsy arising in thalamus, subthalamus, hypothalamus and tegmentum. This classification has not been followed, and the subsequent literature which is reviewed below refers largely to temporal lobe epilepsy, as I shall do in this paper.

B. Occurrence

Temporal lobe epilepsy is stated to be the most common form of epilepsy in adulthood, and the most common of all focal epilepsy. The actual prevalence varies in different reports. Glaser stated the prevalence in children with epilepsy to be at least 25% (11) or 30% (12), although others, including what were probably not cases of temporal lobe epilepsy, reported 11% (13). In adulthood the prevalence of temporal lobe epilepsy is greater, 55% to 65% according to Stevens (14). Gastaut (6) stated figures of 30% in in-patients and 80% in out-patients. However, among 1900 office patients seen by Lennox, 20.7% had psychomotor seizures with or without other types of seizures (1). In 270 female epileptics hospitalized for "inability to support themselves," Margerison and Liddell found that by clinical criteria 78% had temporal lobe epilepsy, by EEG criteria 74%, and by both criteria 61% (15). In an unpublished series referred to in his textbook, Kooi gives figures of 13% psychomotor epilepsy among his clinic population (16).

This large variation reflects sampling of different types of populations, as I have mentioned, and the known variation with age. Thus Lennox's figure, when broken down by age, reveals a history of psychomotor attacks in 10% under 5, 20% from 5 to 19, 24% from 20 to 39, and 34% in

patients 40 years or more. The variation in criteria for diagnosis also makes a valid figure difficult to determine. Patients with psychomotor seizures may also have other types of seizures, and may have demonstrable EEG foci outside the temporal lobes. Gibbs found 450 cases with EEG evidence of focal seizure discharges, of whom 275 had pure spike foci in various cortical areas. 163, or 59%, of these pure foci were in the anterior temporal lobe area; 13% in mid-temporal, 11% in occipital, 10% in parietal and 7% in frontal areas. Psychomotor attacks, occurring in 95% of cases with anterior temporal foci, also occurred in 11% of cases with other foci (17). Patients referred for electroencephalography are already a selected group, and diagnosis is possible on EEG, clinical or combined bases.

As Stevens points out (14), the actual prevalence of temporal lobe epilepsy among epileptics is of importance when considering whether there is any special tendency among them to psychopathology. There does not appear to be a clearly acceptable figure, except to say that it is the most common form of focal epilepsy, and very common among epileptics in general, increasing in incidence with increasing age among all epileptics. This subject will be discussed further in Chapter Six.

C. The Temporal Lobe Seizure

From the time of Jackson there has been general agreement that the temporal lobe seizure incorporates psychomotor symptoms, the automatism, and various psychosensory phenomena including the dreamy state. From a survey of 414 office patients with temporal lobe epilepsy, Lennox (1) outlined the "psychomotor triad," which is a restatement of these components. He gives a distribution of 25% with subjective or psychic phenomena (aphasia, "brown outs," hallucinations, dreamy states, deja vu), 32% with automatic phenomena and impaired contact, and 43% with "psychomotor proper" symptoms (tonic movements, masticatory movements, excessive activity and arrest of activity. There has not always been agreement that the manifestations are indeed ictal, as was described for the dreamy state above. The automatism has been felt to be a post-ictal phenomenon by some, with the preceding psychomotor activity constituting the fit (3). Jackson described it as an event occuring after the paroxysmal discharge, a release from higher control; yet he also considers the dreamy state to occur after these discharges, but to be an epileptic fit even in itself (2). When Liddell isolated 18 patients with automatism from 47 epileptics in a hospital population of 1110 mental patients, he could identify one of the following

before each automatism: grand mal convulsion, adversive attack, or psychomotor activity (twitching, staring, blank expression or rigidity). Thus he believed that the automatism was post-epileptic (3). Feindel and Penfield, finding that behavioral automatism could be initiated by stimulation of periamygdaloid areas in patients with this symptom, noted that the automatism was sometimes accompanied by an aura (abdominal, sensory, cephalic, unreal, tonic or adversive), but sometimes without a "warning" or aura. In the absence of "warning" they consider it ictal; in the setting of such a "warning" they consider it post-ictal (18). These are theoretical difficulties; we really do not know the significance of temporal spiking in the EEG as related to clinical phenomena. The various components of the "triad" appear in a sudden and paroxysmal way, are usually stereotyped for any patient ("his spell"), and may be in any combination. Thus all of the components may be considered part of the temporal lobe seizure, with the electro-clinical correlations as yet undetermined. This is in accord with the descriptions of those authors mentioned below who discuss it.

I shall begin a more detailed description of the temporal lobe seizure with consideration of the automatism. It is a period of altered behavior in which the patient is

only partially able to interact with the environment, rarely able to organize behavior beyond executing learned reaction patterns, and for which he is usually amnesic (19). The antisocial nature of the behavior was mentioned by Jackson and again by Liddell (3); I shall consider it further when discussing ictal rage (see below). Williams, in a speculative re-orientation of temporal lobe function to disturbances of self-concept and integration, described the automatism as:

> Quasi-purposive misbehavior, often of a socially directed sort and often aggressive, too, which is a disturbance of the relationship of the individual to his human environment--- Behavioral disorders of the relationship of the subject to his animate environment. (29)

The frequency of automatism is given in the table below: 48 of 100 temporal lobe epileptics with psychiatric problems (21), 30 of 52 temporal lobe epileptics (22), and 99 cases of integrated, confused behavior in 120 children (11).

The various psychomotor phenomena were listed by Gibbs in 1948: chewing, swallowing, spitting, lip-smacking, rubbing and plucking (7). Lennox described them further:

> Increased tonicity or rigidity of muscles (without clonus), but oftentimes with an adversive movement of the head or eyes or with torsion spasm or---chewing, swallowing

> sucking, smacking or licking the lips, mumbling or drooling, acts called masticatory. Mild cyanosis often accompanies this type of seizure. Posture is usually maintained. Unconsciousness and amnesia are usually complete.

Or,

> Arrest of motion (without muscular tonus), periods of immobility or slumping, with staring, stupor, trance or sleep-like states. Masticatory attacks may attend. Consciousness is impaired or absent. (1)

There is a considerable overlap between these aspects and those commonly called "automatic," but looking only at masticatory attacks, Lennox reports 19% (1), Daly 36% (22), and Glaser 50% (in children) (11), in their respective series.

Although amnesia is usually present for the automatism and psychomotor attack, the visceral, sensory, autonomic, illusory and emotional events that are often present are frequently recalled. These "subjective" components may precede or accompany the other events, or exist by themselves; they may follow the motor attacks by several years (23). Pond describes work by Hill and Mitchell in 1953, analyzing the "aura" in terms of psychopathology:

> The effective component of the aura refers to some early, usually childhood, situation. The perceptual-ideational content achieves its form by the process of symbol formation, displacement, condensation and so on, which are familiar in the formation of dreams and neurotic symptoms. (23)

This relation between the aura and spontaneous dreams is further discussed below considering the hypothesis of Ferguson et al., who reported similar findings in psychotic temporal lobe epileptics (24).

The most common visceral sensation is a rising feeling in the abdomen, also described as constriction or pressure. Analyzing 100 cases, including 69 temporal lobe epileptics, Van Buren found that it generally was "outside normal sensory experience", did not reflect disturbed gastrointestinal function, was referred to midline and epigastrium, and tended to rise if it moved (25). If it rose to the head, it might be associated with feelings of being choked, with nausea or "churning" sensation, or feelings of swelling, pressure, throbbing and heat in the head. It might also be referred to other viscera such as bladder and heart (19).

Somewhat distinct from these sensations are the various autonomic manifestations reported by Gastaut (6) and ascribed to the "hippocampal" variety of psychomotor epilepsy: "abnormal oropharyngeal, esophageal, epigastric, genital, retrosternal and precordial sensations, associated or not associated with nausea, asphyxia or palpitation." In a careful study by Van Buren and Ajmone-Marsan with EEG and monitoring of vascular, respiratory and gastrointestinal function, they observed hypertension, rapid on-off of

tachycardia, low skin resistance, respiratory inhibition in expiration, swallowing movement and inhibition of motility in other gastrointestinal areas, occurring independently of one another and generally not associated with changes in the surface EEG. When bilaterally symmetrical bursts were seen, there was not always autonomic change, and the same burst did not always produce the same change in a given patient. The time sequence was, roughly, early changes in skin resistance and swallowing, early or late respiratory changes, and late tachycardia; at the end of these came the patient's aura or unconsciousness (26,27). The focus was assigned by them to the diencephalon; certainly hypothalamic sympathetic and parasympathetic involvement must occur, as well as that of the several regulatory sites in the brainstem.

Sensory hallucinations occur, and olfactory disturbance has been the hallmark of these attacks for a century. The smell is almost always unpleasant, and when together with the dreamy state was considered an "uncinate fit" (3). This aspect of the temporal lobe seizure was responsible for the confusion in terminology of olfactory brain, rhinencephalon (proper), and limbic system. The anatomical aspects will be discussed. Hallucinations of taste are unusual and difficult to distinguish from smell; when they occur they are more

likely to be acid or bitter (19). Of 100 temporal lobe epileptics, 50 hallucinatory experiences in 45 patients were reported, of which 16 were olfactory and 8 gustatory, frequently both in the same patient (21). Five of 120 children had this olfactory hallucination (11).

As will be apparent from anatomical considerations, visual and auditory hallucinations represent involvement of temporal cortex and white matter, assigned by Gastaut to the temporal variety of psychomotor epilepsy (6). Buzzing, ringing and bells may be heard, sounds may seem louder; others may be seen or heard in strange ways, and lights may appear brighter or appear as flashes (19). 9% in one study (21) and 16% in another (11) suffered from these types of sensory disturbances. 53 of Penfield and Perot's cases, or 10%, had experiential hallucinations, of whom 12 had auditory and 21 visual disturbances (28).

Gastaut (6), Daly (22) and Williams (29) agree that affective disturbances occurring during the temporal lobe seizure may be considered under four categories: fear or terror, depression, pleasure and "unpleasure." To these must be added the feelings of anger or rage, aggression and sexual feelings. Fear or anxiety was the most common, 25 of Daly's 52 patients reporting fear or terror and 14 reporting anxiety (22), fear or anger occurring in 51 of

Glaser's 120 children (11), 10 of Mulder and Daly's 100 cases having dread, fear or terror (21), and 61 of 100 cases with ictal affect reported by Williams having fear or anxiety (29). This is not the natural fear of an impending seizure, but an ictal phenomenon, a fact noted by many. By contrast, pleasure or laughter may be seen as ictal events; the pleasure so intense that, according to Subirana,Dostoevsky (6), was willing to trade ten years of life for one of them. However, as noted by Jackson (2), ictal pleasurable sensations may in time develop into more disagreeable or depressive feeling. Happiness or pleasure was seen in 9 of Williams' cases (29), 12 of Daly's (22), and 5 of Mulder and Daly's (21).

Depression as an ictal event certainly occurs, but in Daly's cases was often prolonged, lasting hours; in Weil's cases, lasting for days (30).

> The reason for this prolongation of the sense of depression is not clear. It may be pointed out that depression normally is a more sustained alteration of affect than is fear, which often is associated with a specific event and hence is more circumscribed. (22)

It has a character varying in these reports from "sweet sorrow" to profound sadness to suicide. Williams states that in 5 of 21 patients with ictal depression, the idea of suicide would arise and was related to the intensity of the depression. One of his patients, whose depression

would last for days, did commit suicide (29). Depression is not uncommon but variable, occurring in 18 of 132 temporal lobe epileptics studied by Weil (30), 21 of 100 cases with ictal affect (29), one case of 100 with psychiatric problems (21), and 5 of Daly's 52 cases. The variability is almost certainly due to difficulty in assessing whether it is ictal or reactive.

Rage or aggression was reported in 5 of Weil's cases (30), 17 of Williams' 100 cases (29), but none of Daly's 52 epileptics (22). Significantly, Glaser reports aggressive activity in 44 of 120 children with limbic epilepsy (11), similar to Ounsted, who found 36 children with outbursts of "catastrophic rage" among 100 children with temporal lobe epilepsy. He states that "the rages which these children show are similar in form to those of normal infants" (37). Glaser states that "emotional turmoil was common, and seizures were precipitated at times of stress or aggravation" (11). I suspect that this accounts for a higher frequency of ictal rage and aggression in children. Isolated case reports of ictal rage exist, such as the case of a 19 year old boy who would try to strangle his mother, crush his brother, attack or threaten doctors and nurses or try to commit suicide, rendered a case of the Kluver-Bucy syndrome by Terzian and Dalle Ore (31). Another case reported by Ervin et al. (32),

a 20 year old woman, had ictal rage in which she had smothered a 26 month old baby and stabbed two people; depth EEG revealed seizure activity from the hippocampus, and stimulation induced a fit of rage. However the case reported by Brewer (33) of a 24 year old man who murdered two people and was subsequently aphasic, confused and psychotic, was probably not a case of temporal lobe epilepsy as claimed. Although there was dilatation of the temporal horns of the lateral ventricles, there was no EEG evidence of seizure activity, nor was there any previous history of other seizures.

Sexual sensation is rare in the seizure, and of two forms, somatic genital sensation (tingling, paresthesias) and erotic affective experience. Daly reports having seen only two cases of the former and one of the latter (22). Currier et al. reported two cases, a 52 year old woman who displayed pelvic movements and vocalization appropriate to sexual excitement, and a 37 year old woman who showed arm movements that were interpretated as masturbatory (34). Properly speaking these are sexual automatisms and not erotic experience. Bancaud et al. reported a case of a 20 year old man whose paroxysmal sexual experience was intense and often seemed orgasmic (35,36); he found only 36 cases in a review of the literature.

Ictal events that may be grouped as perceptual illusions include a variety of unusual experiences. One is the dreamy state, described above, generally referred to as the feeling of familiarity or deja vu; feeling of unfamiliarity also occurs, jamais vu, and may or may not arouse anxiety. Feelings of strangeness or unreality occur, described as being vacant, lost, out of this world, and even very common objects are referred to as strange. Forced thinking may occur, in which an idea supplants all others for the duration of the seizure. A sort of "panoramic" memory may occur in which a past event is not so much remembered as relived, comprehensively, in all its emotional intensity. Some more complex visual or auditory hallucinations may approach these experiences in elaborateness (19). The occurrence of these events taken together was 37% in Mulder and Daly's review (21), and 40% in Daly's (22).

Another, less common perceptual illusion is one of distortion of the body image, so that the patient sees himself in unusual ways. Ionasescu describes six such cases, found among 80 cases of temporal lobe epilepsy. The illusions included feelings that parts of the body such as tongue, ear or head were bigger, that the patient was lifted into the air or floating, and feeling that the patient was not himself but rather someone else was him (39). These feelings

of partial or complete depersonalization are also reported by Williams (20).

It is readily apparent that a wide variety of events may characterize the temporal lobe seizure, particularly when it is recalled that any combination of all of these may be seen in a given patient. I have outlined them below.

A. Motor activity
    1. Simple
        a. Masticatory attack
        b. Excessive activity
        c. Increased tone
        d. Arrest of activity
    2. Complex
        a. Confused automatism with amnesia
        b. Integrated automatism with amnesia
    3. Visceral (autonomic)
B. Sensory experience
    1. Hallucination
        a. Simple sensory experience
            (1). abdominal, visceral
            (2). tingling, paresthesia
        b. Special sensory experience
            (1). olfactory
            (2). gustatory
            (3). auditory
                (a). simple sounds (buzz, ring)
                (b). complex sounds (macroacusia, microacusia)
            (4). visual
                (a). simple (flash, bright light)
                (b). complex (micropsia, macropsia, metamorphopsia)
    2. Illusion of body image
        a. partial (limb, head)
        b. complete
            (1). Illusion of position
            (2). Depersonalization
    3. Illusion of unreality or strangeness
C. Orientation to time
    1. Arrest or rapidity of time
    2. Recall

a. Forced memory or thinking
b. Reliving memory (panormaic memory)

3. Dreamy state
a. Deja vu
b. Jamais vu

D. Emotional experience
1. Fear, anxiety, dread, terror
2. Depression, suicide
3. Pleasure
4. Rage, anger, aggressive
5. Sexual

This outline of events occurring in temporal lobe seizures is a composite of several, especially Williams (20), Schmidt and Wilder (19) and Gastaut (6), with the exposition above.

The following table is a comparative summary of the frequency of some of the events described:

| Event | Weil (30) | Williams (20) | Mulder & Daly (21) | Daly (22) | Glaser (11) |
|---|---|---|---|---|---|
| Automatism | | | 48 | 30 | 99 |
| Visceral | 28 | | taken both together, | 20 | 64 |
| Autonomic | | | 54 cases | 35 | 49 |
| Olfactory | 9 | | 16 | 6 | 5 |
| Gustatory | 2 | | 8 | 4 | |
| Auditory | | | 9 | 2 | taken both together, |
| Visual | | | 18 | 9 | 20 cases |
| Fear, Anxiety | 16 | 61 | 10 | 39 | 51 |
| Pleasure | 0 | 9 | 5 | 12 | |
| Depression | 18 | 21 | 1 | 5 | |
| Rage,Anger | 5 | 17 | 1 | | |
| "Unreal" experience | 7 | | 37 | 20 | |
| N (total cases) | 132 | 100 | 100 | 52 | 120 |

In 1964 Gastaut proposed for the Commission on Terminology of the International League Against Epilepsy the following new classification of epileptic seizures (38):

1. Partial seizures
   A. Elemental symptomatology
      (1). Motor symptoms
         (a). Focal motor
         (b). Jacksonian
         (c). adversive
         (d). postural

(e). somatic inhibitory
(f). involving speech
(2). Special sensory or somatosensory symptoms
(a). somatosensory
(b). visual
(c). auditory
(d). olfactory
(e). gustatory
(f). vertiginous
(3). Autonomic symptoms
B. Complex symptomatology (which may sometimes begin as elemental symptomatology)
(1). Impaired consciousness alone
(2). Intellectual symptomatology
(a). dysmnestic disturbances (including amnesia, deja vu)
(b). ideational disturbances (including "forced thinking")
(3). Affective symptomatology
(4). Psychosensory symptomatology (illusions, hallucinations)
(5). Psychomotor symptomatology (automatism)
(6). Compound forms
C. Secondarily generalized (symmetrical or asymmetrical, tonic or clonic, most often tonic-clonic)
2. Generalized seizures
A. Non-convulsive
(1). Impaired consciousness alone (absence)
(2). Associated with other phenomena
(a). myoclonic absence
(b). increased tone
(c). diminished tone or absent tone
(d). automatism
(e). autonomic phenomena (abdominal seizures, etc.)
B. Convulsive
(1). Myoclonic jerk
(2). Clonic
(3). Tonic
(4). Tonic-clonic
3. Unilateral seizures in children
4. Erratic seizures in new-born
5. Unclassified

It will be readily seen that symptoms described in much of section 1 and especially section 1.B. are seen in the entity of temporal lobe epilepsy as usually described. Because of this frequent overlap and combination of symptoms, and the lack of adequate anatomical-clinical correlation, I find it more fruitful to continue to refer to the temporal lobe seizure.

In 1969 Gastaut attempted to classify epilepsies as he had seizures, and proposed the following:

1. Partial or focal epilepsies
   - A. With elemental symptomatology
     - (1). Motor symptoms
     - (2). Sensory symptoms
     - (3). Autonomic symptoms
   - B. With complex symptomatology
     - (1). Mental symptoms
     - (2). Psychosensory symptoms
     - (3). Psychomotor symptoms
2. Generalized epilepsies
   - A. Primary (cryptogenic)
     - (1). Absence
     - (2). Myoclonus
     - (3). Tonic-clonic
   - B. Secondary (symptomatic)
3. Unclassifiable epilepsies

He added that, if an anatomical localization were possible for the partial epilepsies, it would not be incorrect to refer to them as such, e.g. "temporal partial epilepsy"(40). Commenting on this proposal, Masland pointed out that classification was possible on the basis of the etiology, seizure pattern, electroencephalogram, anatomy, age and any precipitating patterns or "reflexes" (41).

In this paper, I shall refer to "temporal lobe epilepsy" as that entity called "partial complex epilepsy" above, because this is consistent with trhe general understanding of the term and is in more general usage. The other descriptive factors will be discussed for this entity of temporal lobe epilepsy.

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## Chapter Two

## BIOLOGY

### A. Anatomy

Inasmuch as an appreciation of neuroanatomy is essential to understanding neurological phenomena, I shall attempt to present here in summary form well-established aspects of the anatomy of the temporal lobe, the limbic structures and their connections.

1. Temporal Cortex (Neopallium)--The anatomical divisions of the cortex are: the transverse gyri (of Heschl), Brodmann areas 41,42; the superior temporal gyrus, Brodmann area 21; the inferior temporal gyrus, Brodmann area 20; the fusiform gyrus, Brodmann areas 35,36. Brodmann area 38 encompasses the anterior temporal tip. Areas 41 and 42 are auditory sensory areas receiving fibers from the medial geniculate body and transmitting sounds to be differentiated for loudness, quality and pitch. Area 41 is primarily receptive, and area 42, which overlaps onto the superior temporal gyrus, is primarily associative for auditory stimuli. Each cochlea is represented in both hemispheres. Electrical stimulation produces elementary tones with no complex or

psychic quality. Area 22, which receives fibers from areas 41 and 42, is implicated in the interpretation of sounds and their meaning; association of sounds with past experiences is a function of this area. Lesions in this area may cause "word deafness" or auditory sensory aphasia (1,2).

2. Temporal White Matter--Association fibers connecting the temporal cortical auditory representations pass through the temporal lobe, corpus callosum and trapezoid body. Other temporal association pathways pass through the posterior part of the anterior commissure. Afferent fibers arrive from the cochlea via the medial geniculate body (the auditory radiation); efferents ramify widely in the cortex of other areas of the brain. A portion of the optic radiation passes through temporal lobe areas. In its passage from lateral geniculate body to calcarine cortex, the optic radiation can be separated into dorsal and ventral geniculocalcarine tracts. The ventral fibers, arising from the most lateral portion of the lateral geniculate and representing the upper quadrants of the visual field, pass rostrally and anteriorly deep into the temporal lobes, turn caudally around the lateral ventricle and pass along the inferior horn back toward the calcarine cortex. Dorsal geniculocalcarine fibers pass more directly to occipital lobes and not through temporal areas (2).

3. Septum--The septal cortical area is located anterior to the rostrum of the corpus callosum and rostral to the anterior commissure. It is composed of the subcallosal area and the paraterminal gyrus. The septal subcortical nuclei, located rostral to the anterior commissure, interconnect with both hippocampal and amygdaloid fiber systems. The medial septal nucleus receives afferents from the fornix and the amygdaloid complex; it becomes continuous with the diagonal band and connects to the amygdala. It also receives afferents from the reticular formation of the midbrain tegmentum. Both medial and lateral septal nuclei send efferents to the habenular nucleus and then to midbrain tegmentum and lateral hypothalamus, via the striae medullaris and fasciculus retroflexus. The medial forebrain bundle also carries efferents to midbrain and hypothalamus (2).

4. Olfactory Pathways--Fibers leaving the olfactory bulb divide into medial and lateral olfactory stria. Medial olfactory fibers become continuous with the septal cortical areas. Lateral olfactory fibers terminate in the prepyriform and periamygdaloid areas, from which efferents are sent to amygdala, thalamus, hypothalamus, anterior parahippocampal gyrus and uncus. Fibers from the parahippocampal gyrus and uncus are sent to the hippocampus, insula and frontal cortex (2).

5. Hippocampus--A continuous body of phylogenetically old cortex (archipallium) develops, with the development of the temporal lobes, into an arc-shaped entity, whose "proximal" portion is poorly developed because of the massive presence of the corpus callosum. This portion, the indusium griseum, is found between corpus callosum and cingulate gyrus. The "distal" portion follows the lateral ventricle temporally and differiates into the hippocampal formation (2).

The embryological hippocampal fissure, whose appearance "signals the first attempt at cortical maturation"(3), pushes inward into the inferior horn of the lateral ventricle; the cortex thus invaginated becomes the hippocampus proper. The medial lip of the fissure becomes the dentate gyrus and the lateral lip becomes the parahippocampal gyrus. The dentate gyrus extends caudally with the fimbriae to the splenium of the corpus callosum, where it separates from them to connect to the indusium griseum. Rostrally, it passes forward to connect to the uncus. The parahippocampal gyrus is continuous caudally with the isthsmus and the cingulate gyrus (2).

Afferents to the hippocampus come from anterior temporal cortex, from the cingulate gyrus, from the olfactory bulb, and from the medial forebrain bundle via the septal nuclei (4). There are various complex connections between

the hippocampi (4). Efferents from the hippocampus traverse as the fimbriae back to the splenium of the corpus callosum, then turn beneath it to pass rostrally as the fornix. The anterior commissure divides fornix fibers, precommissural fibers going to septum and lateral and anterior hypothalamus, postcommissural fibers going to the mammillary bodies and also directly to the thalamus. Via these hypothalamic structures there are connections to the midbrain tegmentum and (through the mammillothalamic tract) to the thalamus, which projects to the cerebral neocortex (2).

It is generally accepted that the hippocampus has a lower threshhold for seizure initiation than most other cortical structures (4,6,7), and this is advanced as an explanation for the cause and frequency of temporal lobe seizures. Injuries to this structure are certainly seen, but it is difficult to say whether they are cause or effect of the seizures (see below) (4). In an extensive review on the hippocampus, Green states:

> The hippocampus has a very low seizure threshhold in man and animals. Seizural discharges initiated in one part of the rhinencephalon such as the hippocampus readily spread to other adjacent rhinencephalic areas and to the hypothalamus or become generalized. (4)

6. Amygdala--The amygdaloid nuclear complex, which is properly one of the basal ganglia of the brain, is located

internal to the uncus, and may be considered the phylogenically oldest part of the basal ganglia. It is divided into groups, the corticomedial nuclear group and the basolateral nuclear group. The corticomedial group is less well developed, and is concerned largely with reception of fibers from olfactory nuclei and projection of fibers to septum, hypothalamus and brainstem. The basolateral group receives olfactory fibers that have been relayed through prepyriform cortex (see above), and also projections of other visceral and sensory systems. One efferent pathway is through the striae terminalis to anterior, preoptic and ventromedial hypothalamic areas. Another is through the ventral amygdalofugal projection to dorsal and lateral hypothalamic areas, septum, dorsomedial thalamus and cortex. The cortex receiving amygdala afferents includes the septal cortical area, the cingulate gyrus rostrally, the several temporal gyri, the insula and orbitofrontal cortex (2). "Fairly" direct connections between amygdala and hippocampus exist, but it is not clear whether these are monosynaptic or polysynaptic (2,4).

7. The Limbic System--The name "limbic system" was coined by Broca in the nineteenth century; it is taken to include olfactory bulb and tracts, septum, hippocampus, amygdala, cingulate and parahippocampal gyri. Initially it was believed to subserve olfactory functions, hence the name rhinencephalon,

but this is no longer held. MacLean and others have divided the limbic system into two concentric rings, the inner one comprising septum, indusium griseum, hippocampus and corticomedial amygdaloid nuclei, and the outer one comprising orbitofrontal cortex, cingulate gyrus, isthsmus, parahippocampal gyrus, basolateral amygdaloid nuclei, uncus and olfactory bulb. The outer ring connects to the inner ring and from there to the structures outlined above (3,5). The functions of these structures are nowhere near as clearly known as the anatomy, and will be discussed next. Figure 1 attempts to depict the location of these areas of the brain.

B. Functions

As mentioned above, much of the work on function of the temporal lobes relates to a theory of emotion first elaborated by Papez in 1937, when he proposed the following idea:

> The central emotive process of cortical origin may then be conceived as being built up in the hippocampal formation and as being transferred to the mammillary body and thence through the anterior thalamic nuclei to the cortex of the gyrus cinguli. The cortex of the cingular gyrus may be looked on as the receptive region for the experiencing of emotion as the result of impulses coming from the hypothalamic region---Radiation of the emotive process from the gyrus cinguli to other regions in the cerebral cortex would add emotional coloring to psychic processes occurring elsewhere. This circuit would explain how emotions may arise in two ways: as a result of psychic activity and as a consequence of hypothalamic activity. (8)

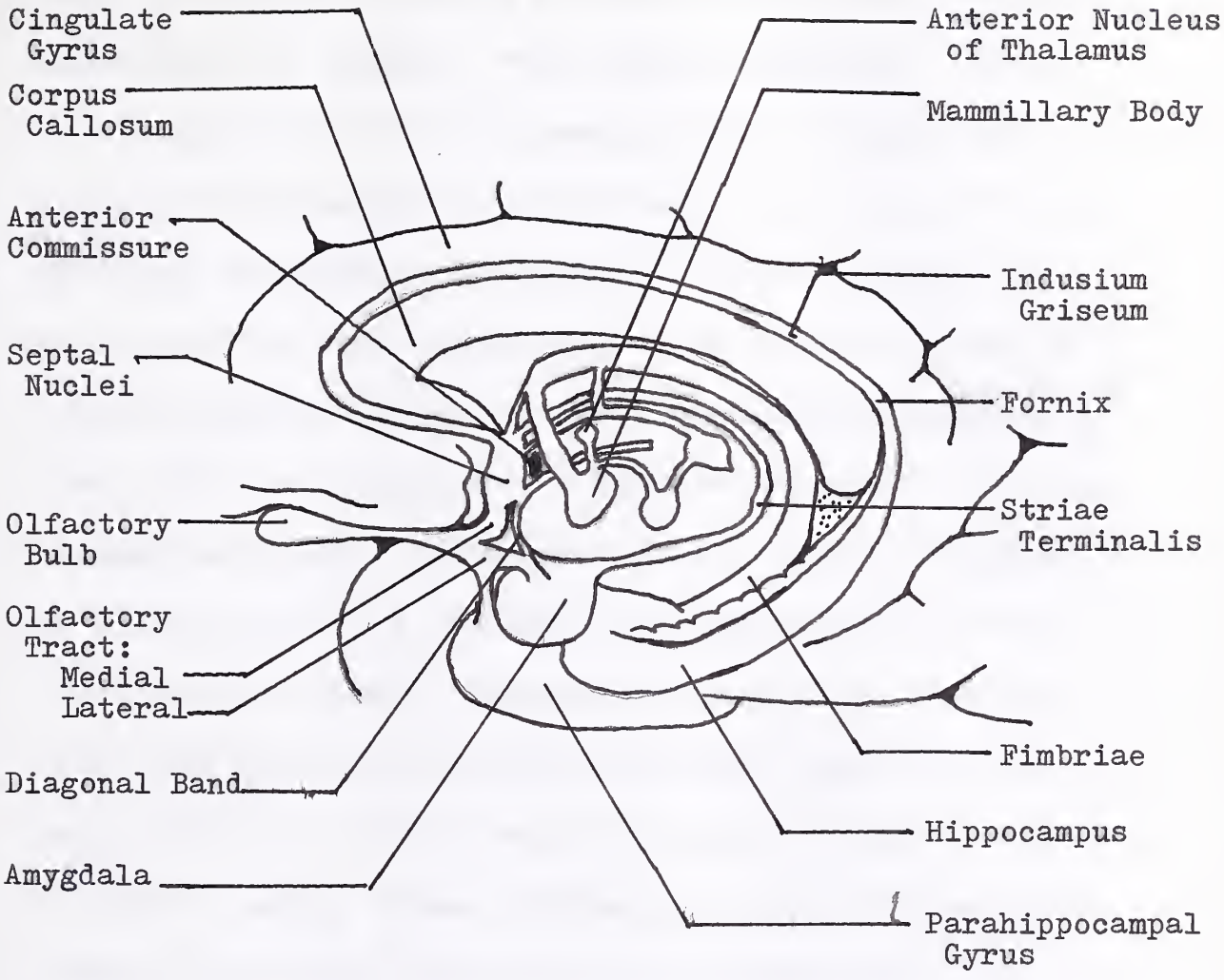


Figure 1
Anatomy of The Limbic System

From Truex and Carpenter, p. 522

Papez no sooner proposed this mechanism than Klüver and Bucy published the results of the first of their ablation experiments in monkeys. They found no behavioral effect from removal of the first temporal gyrus bilaterally, removal of the second and third temporal gyri bilaterally, bilateral extirpation of prefrontal areas or removal of the second and third temporal gyri on one side. When an entire temporal lobe was removed, the animals seemed more tame. But when both temporal lobes were removed, striking changes were seen. The animals showed "psychic blindness" or visual agnosia, a tendency to attend to and react to every visual stimulus ("hypermetamorphosis"), compulsive oral investigation of objects, increased sexual activity and a failure to exhibit motor or vocal reactions indicative of fear or anger. These findings could not be generated simply by bilateral severing of the connections of the temporal lobe to frontal or parietal lobes (9). In experiments involving ablation of somewhat different areas, orbital and insular cortex in addition but not including the hippocampus, the changes were much less severe: there was no change in visual, motor, or tactile behavior, but alteration in taste, energy metabolism and reaction to stimuli were noted. More restricted lesions led to subdivision of function thus: locomotor activity in the frontal areas,

taste in the insular areas, and food intake and temperature regulation in the temporal pole and amygdala (10). The syndrome found by Klüver and Bucy has also been seen in humans suffering the same surgery. Thus Terzian and Dalle Ore reported the case of a 19 year old boy with intractable and violent seizures, and interludes of several weeks in which he was normal and pleasant. No improvement was seen after unilateral temporal lobectomy, but after bilateral lobectomy he was strikingly different. He was unable to experience any emotion, not rage or love, and showed no evidence of emotional involvement with anything or anyone, even his parents. Objects and symbols were emotionally indifferent. He was not aphasic, but his symbolic language deteriorated so that he expressed little and perseverated markedly. He had a severe memory loss, could remember nothing of the past or plan the future, and was unable to understand the meaning of words. He was seen to approach everyone, possess all objects around him, and showed hyperphagia, eating anything put in front of him. After a few weeks he became hypersexual, with exhibitionism, masturbation and homosexual interest. This complete psychic destruction "cured" his temporal lobe seizures, but his convulsions subsequently recurred (11).

More specific ablations have demonstrated more

restricted changes. In 1947 Bard and Mountcastle published their well-known account of "sham rage" in cats after amygdalectomy with or without accompanying removal of temporal isocortex. The cats were irritable, savage and easily provoked to a rage reaction termed "sham" because of its inappropriateness. Amygdalectomy in other hands and in other animal species have not confirmed this finding, and in fact have led to placid and docile behavior rather than rage (2,30). Narabayashi et al. removed the amygdala unilaterally or bilaterally in 60 patients with severe behavior disorders, 46 of whom were epileptic (mostly grand mal, some psychomotor). In 51 cases there was marked reduction in emotional excitability and "normalization" of their social behavior and adaptation. In no cases were signs or symptoms of the Klüver-Bucy syndrome seen (12). In monkeys having bilateral amygdalectomy, temporary changes in behavior were seen, characterized by tameness, fearlessness, asocial behavior and diminished sexuality; by the fourth or fifth month after surgery, behavior had reverted to that seen before surgery (13). Removal of the hippocampus bilaterally in nine patients studied before and after surgery with formal testing of memory and intelligence demonstrated a "persistent impairment of recent memory whenever the removal is carried far enough posteriorly to damage portions

of anterior hippocampus and hippocampal gyrus." After these medial temporal ablations, no changes in intelligence, personality or behavior suggestive of the Klüver-Bucy syndrome were seen (14). This and other work seemed to localize placidity and docility to pure amygdalectomy and hypersexuality to additional involvement of the peri-amygdaloid pyriform cortex (2).

It is interesting that one of Revitch's cases demonstrated several signs of the Klüver-Bucy syndrome without ever undergoing temporal lobe surgery. An eight year old girl with a history of high fever at one year, minor seizures beginning four months later, and grand mal convulsions beginning two years after this, she was prone to frequent convulsions, several a day and three or four at night. At age eight, although evidently retarded, she was seen to have lost fear of pain or danger and was compulsively oral, ingesting feces and bits of glass (41). It is possible that in this case progressive hypoxic damage to temporal and limbic areas achieved a "functional ablation" of these structures without causing typical temporal lobe seizures.

As noted above, electrical stimulation in the area of the amygdala in temporal lobe epileptics by Van Buren resulted in clinical automatism, accompanied by the

autonomic changes of hypertension, tachycardia, fall in skin resistance, esophageal peristalsis, defecation, salivation, piloerection, hypothermia or hyperthermia, and inhibition of respiratory rate, rhythm and amplitude (15). A woman not previously epileptic showed epileptic activity in the amygdala and convulsion when during a structured interview with depth EEG recording the subject of anger at her father for incestuous desire was considered (16). Masticatory phenomena, adversive reactions, fear and rage, confusion, disorientation and amnesia have all been observed during electrical stimulation of the amygdala (2). Many of these automatic and autonomic reactions are also seen in electrical stimulation of cingulate and orbito-frontal cortex in animals (2).

The late Lord Brain, in an article written shortly before his death, wrote of the "neurology of memory" thus:

> Part of the hippocampal gyrus appears to be the start of a pathway through which current experiences are converted into memories. This pathway appears to run from the hippocampal gyri through the fornices to the mammillary bodies and thence to the thalami and cingulate gyri. It is not suggested that these parts of the brain are concerned with the storage of memory as such: they are rather the route by which experiences are processed or conducted to the areas in which they are ultimately stored. Destruction of the hippocampi therefore leads to loss of the capacity to convert current experiences into memories (17).

Stimulation of the hippocampus was found to generate reminiscent reactions, older memories with deeper stimulation. The recall was always associated with appropriate emotional coloring (18). Bilateral hippocampal stimulation in another study was seen to produce clouding of consciousness and amnesia for remote events; stimulation of the amygdala produced re-experiencing of the past (19). Green wrote that he was "unimpressed" with the attempt to localize the memory itself in the hippocampus, feeling that the amnesia seen in these studies was more likely due to disruption of the sort of "transactional mechanisms" described by Brain (4).

More complex "experiential" hallucinations are also seen with stimulation of the temporal lobes. Mahl et al. reported illusional, ego-alien experiences with anterior temporal stimulation in temporal lobe epileptics, which seemed to them to reflect recent experience and recent mental content. They hypothesized a shift from secondary-process to primary-process organization (dream-like) (20). Describing induced hallucinations into experiential and interpretative, Penfield considered the former a hallucination of past experience and the latter an illusion of present experience (4,21). Because it is possible to elicit these very specific previous experiences by temporal cortical

stimulation, Penfield feels memory is stored here (22), but others feel memory is diffusely located in the brain (4).

It is impossible to be specific about the localization of function in the brain. There is much overlap between functions and areas studied, and experiments on one area may reveal behavioral changes due to involvement of quite separate but connected areas of brain. Allowing for the possibility of the same area serving different functions in man and in research animals, there still emerges from the many experiments and reports this sense of overlap. I have only attempted to sketch a picture of these studies with the selections discussed above. I think that the many connections and interrelations between these structures accounts for some of this lack of specificity seen. It may well be that in the intact man or laboratory animal the variety of events seen in experiments and in spontaneous temporal lobe seizures are in fact the result of very elaborate interactions between many parts of the brain separated by anatomical studies.

C. Pathology

Softening of the hippocampus was noted over a century ago, but it was Sommer who noted the loss of pyramidal cells in the H-1 sector (or Sommer's sector) of the hippocampus (23). The significance of this was not apparent until later work connected dreamy states, psychomotor attacks and the mesial temporal structures; thus Penfield is reported to have said in 1929:

> The changes in Ammon's horn (reported by Spielmeyer) are perhaps the farthest removed from the attention of those interested in the epileptic mechanism (23).

The relevance of this area soon became apparent through the electroencephalographic work of the Gibbs' and the surgical experience of Penfield himself.

In 1953 Earle, Baldwin and Penfield reported the pathology they had observed in resected specimens of temporal lobe in temporal lobe epilepsy. In 100 of 157 cases there were "ischemic" changes in the hippocampus and adjacent areas: the uncus, hippocampal gyrus and first temporal gyrus were most frequently involved in atrophy. The areas involved were shrunken, yellow, avascular, with increased fibrous astrocytes and scattered ganglionic destruction. The combination of gliosis and neuronal destruction in hippocampus and adjacent areas they termed "incisural sclerosis" (24). Others found this lesion, some

believing it the cause of "secondary" epilepsy (as opposed to "primary" or tumor-related), and as such the most common form of temporal lobe epilepsy among institutionalized epileptics (23).

In 1964 Falconer, Serafetinedes and Corsellis reported the pathological findings in 100 cases of temporal lobe epilepsy undergoing removal en bloc of the temporal lobe. They found gliosis and neuronal loss in Sommer's sector of the hippocampus and in amygdala; when more severe, they also found damage in other parts of hippocampus, amygdala and uncus, and in the most severe cases, also in the cortex of the hippocampal, fusiform and temporal gyri, particularly the second and third layers of the cortex. This lesion was seen in 53 of their 100 cases. Because of inferences about the etiology (see below), they rejected the term "incisural sclerosis" and proposed "mesial temporal sclerosis" for the findings in this relatively diffuse injury (25).

The lesion described is not completely accepted, partly because of the argument over etiology, partly because of its presence in non-epileptics and epileptics without temporal lobe seizures (6). However, it is seen in between 50% and 75% of temporal lobe epileptics who come to surgery for lobectomy (23-25).

Other pathology seen in the temporal lobes includes small scars and "infarcts", hamartomas, "equivocal" changes, and tumors, benign and malignant (23,25). These will be considered below.

D. Etiology

1. Tumors--Brain tumor is not a frequent cause of epilepsy in unselected populations, but it is frequently associated with epilepsy when it does occur (6). They are said to cause seizures in from 35% to 60% of cases, the nearer the tumor to cortex, the more likely a seizure (26). In a review of 898 cases of tumors specifically in the temporal lobe, drawn from the literature prior to 1953 and from their own experience, Strobos reported seizures in general to occur in an average of 49% of cases. Dreamy states alone occurred in 18%, olfactory and gustatory hallucinations in 15%, auditory hallucinations in 1%, and visual hallucinations in 8%. Grand mal seizures occurred in 24% (27). A recent study of 308 cases of temporal lobe tumors found epilepsy in 41% of intracerebral and 21% of extracerebral tumors; it was the initial symptom in 19% and an early symptom in 39% (28).

In series encompassing epileptics studied for cause of epilepsy, tumor is not usual. Such series may be biased, e.g., patients with symptoms of tumor may never come to a neurologist for management (6). In a review of seven such series, the frequency of tumor in non-focal seizures in adults was given as 10%, and in focal seizures as 30-40% (6). Among 88 patients with temporal lobe epilepsy

referred to a surgeon, 15 neoplasms were found (17%) (23). 24 tumors of the temporal lobe were found among 100 patients with temporal lobe epilepsy seen by neurologists (29). In another such series of 52 patients, tumor was found in 6 (12%) (30).

The later the onset of seizures, the more likely is the cause to be tumor. Reasons given for this include the relatively greater number of children with onset of seizures, the greater proportion of "symptomatic" seizures in adults, and the greater number of children with epilepsy (6,26).

2. Infection--Gastaut reports encephalitis as the cause of temporal lobe epilepsy in 25% of Gibbs' cases and 20% of his own (31). Again, seizures are more likely to the extent the infection (encephalitis or abscess) involves the cortex (26). Herpes simplex (Herpesvirus hominis) encephalitis is known to cause personality changes, bizarre behavior and hallucinations and to localize in the temporal lobe (26), and the question of mild herpes infections causing temporal lobe seizures has been raised recently (32-34).

3. Trauma--Trauma was given as the cause of temporal lobe seizures in 314 of 2485 cases studied by Gibbs, or 17% (6). The nature of the head injury makes a difference, as closed head trauma or concussion results in seizures in only one to five percent, whereas penetrating injury may cause

seizures in 20% to 40% (6,26). Gastaut felt that the fact that 80% of his cases of temporal lobe epilepsy had onset before thirty years old correlated well with the fact that in 50% of the total cases cranial injury was given as the cause (31). By contrast, Lennox felt that because the frequency of his patients with epilepsy having temporal lobe epilepsy increased with age, these patients had epilepsy as a consequence of brain damage (35). In both situations the likelihood of birth injury, as opposed to later trauma to the brain, is not separated as a factor. The significance of this differentiation will be apparent in the discussion of mesial temporal sclerosis (see below).

4. Mesial Temporal Sclerosis--I have selected to use this term for the pathology discussed above because it does not convey bias about etiology as does "incisural sclerosis".

Wilder Penfield noted that in many of his patients for temporal lobectomy for temporal lobe epilepsy, there was a history of brain injury in birth or infancy. He noted further that the vascular anatomy of the area of the mesial temporal lobe is such that the anterior choroidal artery, branches of the middle cerebral artery and branches of the posterior cerebral artery, all of which supply these temporal structures, must cross the free edge of the incisure of the tentorium to reach the temporal lobe. The lesions

seen looked ischemic and avascular, and he proposed that compression of these vessels at birth damaged the structures supplied, which then over several years "ripened" into an epileptogenic focus (24). Earle, Baldwin and Penfield reported the results of experiments in which the heads of stillborn babies were compressed with rubber tubing, frozen and sectioned; herniation was indeed seen (24). Herniation was uncommon in autopsy of children with difficult birth, a fact attributed to the elasticity of the head and brain, and necessitating freezing. The role of the freezing in causing herniation, and the nature of the reproduction of birth trauma by compression, make these experiments difficult to interpret. Only 25 of their 100 cases of "incisural sclerosis" gave a history of difficult birth, 49 being cases of postnatal head injury and 26 cases of infantile brain infection (24). Thus the argument that vascular compromise at birth due to herniation from difficult delivery is the cause of mesial temporal sclerosis in temporal lobe epilepsy is difficult to accept.

Murray Falconer found mesial temporal sclerosis in 53 of 100 cases of temporal lobectomy for temporal lobe epilepsy in which the lobe was removed as a block. In 28 of these cases onset of seizures was at less than ten years of age. Thirty to forty percent gave a history of infantile

convulsions, which history was uncommon in groups of other pathology (25,36). Thus he wrote:

> The most common cause of mesial temporal sclerosis is an asphyxial episode, such as a severe febrile convulsion occurring between the ages of six months and four years, at a time of life when the mesial temporal structures---are particularly vulnerable to anoxia. The nerve cell damage then sustained "ripens" into a sclerotic process---which becomes a potent epileptogenic lesion in its own right. (37)

The general occurrence of febrile convulsions in children is about five percent (6).

In general support of Falconer's thesis is a recent experiment in which lesions of "ammon's horn sclerosis" were produced in hyperthermic guinea pigs after repetitive convulsions induced by hypoxia or hypoglycemia (38). In rebuttal, again only a minority of the cases seen gave a history consistent with the thesis of convulsions as the cause of their temporal lobe epilepsy. Lennox's study is pertinent, in that comparing the occurrence of grand mal seizures in patients with temporal lobe epilepsy with the age distribution of these patients, it was seen that in the age groups having the greatest frequency of temporal lobe seizures there was also the greatest frequency of previous grand mal convulsions (35). However, as this was also the oldest age group, the simple effect of time may play a role.

In summary, the etiology of mesial temporal sclerosis must be considered unknown. Neither argument presented above can account for all the observed phenomena. It may be that the character common to both, the role of hypoxia, is a "common pathway" for damage of diverse origin, which acts on the structures to generate the lesion seen. Thus in the 73 of 88 cases for lobectomy in temporal lobe epilepsy and not secondary to tumor, Green found a history of difficult birth in 22, post-natal trauma in 21, and febrile convulsions (including those due to brain infection) in 14 (23).

5. Other Causes--Lesions found in temporal lobe epilepsy also include small hamartomatous growths, meningo-cortical cicatrices, vascular malformations and hemangiomas (23,25,36). Agents more often causing generalized convulsions include alcohol (39), drug withdrawal (40), surgery, metabolic and genetic diseases (6).

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## Chapter Three

## INHERITANCE AND COMPLICATIONS

### A. Inheritance

The determination of inheritance depends in part on the general prevalence of epilepsy in the population, as a point of comparison. Draft induction records of the world wars gave a figure of 0.5% (1), somewhat higher than that found by Kurland in Rochester, Minnesota, 0.37% using strict criteria (23), and that of Logan and Cushion in Britain, 0.33% also using strict diagnostic criteria (24). Gudmundsson obtained a similar figure in Iceland, 0.35% (25), and this appears to be a reliable consensus prevalence.

Metrakos and Metrakos have separated thinking on genetics in epilepsy into two "schools", those of Lennox and Ahlström (1). In 1951 Lennox reported that examining the parents, siblings and children of 4231 epileptics seen in his office, and comparing prevalence of epilepsy to that found among draftees in World Wars I and II,0.5% (1), the prevalence in relatives of patients with symptomatic epilepsy was 3.6 times that expected, and in relatives of patients

with essential (idiopathic) epilepsy it was 7.2 times that expected (2). The overall prevalence was 3.2%, but when the epilepsy began in infancy it was 7.6%, and when it began after age 30 it was 1.5% (2). The occurrence of epilepsy in near relatives of patients with psychomotor seizures alone was 2.6%, and in relatives of patients with psychomotor and grand mal seizures it was 2.8% (3). Ahlström, examining relatives of patients somewhat older than Lennox's, found an overall prevalence of only 1.5%, but again less in the symptomatic group (4). Metrakos and Metrakos point out that Ahlström's paper also argues against social prohibitions against marriage for epileptics (1).

Results of concordance between monozygotic and dizygotic twins for epilepsy support a genetic component: in one study, 85% in monozygotic twins without prior brain damage, 27% with prior brain damage; and 16% and 13% in dizygotic twins, respectively. Another study found concordance for epilepsy in 37% of monozygotic pairs and 4% in dizygotic pairs. A smaller study found concordance in 14 of 26 monozygotic pairs and in 1 of 7 dizygotic pairs (5).

The frequency of a family history of epilepsy in patients with temporal lobe epilepsy varies in several studies. As mentioned, Lennox found 2.8% (3). Gibbs reported 4.5% of his 300 cases of psychomotor discharge on

EEG had a family history of epilepsy (6). Glaser found a family history of convulsions in 11 of 120 children with limbic epilepsy (7), but Holowach et al. reported a family history in 55 of 100 children with psychomotor seizures (8). Currie et al. found 11% of 666 temporal lobe epileptics to have a family history of epilepsy (26). The variation with age of family history of epilepsy, mentioned above, may underlie these differences.

Two studies comparing EEG in families of patients with temporal lobe epilepsy and matched controls found in one instance that in half the cases of temporal sharp waves or psychomotor seizures, the patient's mother also had an abnormal EEG (9). Bray and Wiser found at least one family member with a temporal EEG abnormality in 12 of 40 families of patients with temporal lobe epilepsy, and in 2 of 40 families of normal controls. When seen, the abnormality was twice as likely to be in siblings or children as in the parents of the patient (10).

It appears possible to conclude from this that:

1. An inherited predisposition to seizures exists, but varies in degree.
2. A genetic cause is most likely in epilepsy of childhood onset, and least likely in epilepsy of late onset or due to focal brain injury.

3. The risk decreases with decreasing degree of genetic relationship.

These conclusions (5) appear valid for temporal lobe epilepsy as well as for all epilepsy.

B. Temporal Lobe Epilepsy in Childhood

There are several aspects of the process which are somewhat different in chhldhood. The likelihood of a genetic etiology has been mentioned, and the likelihood of a history of perinatal difficulty is also greater, 32 of 120 in one study (7), 51 of 125 in another (11). Neurological diseases such as tuberous sclerosis and Sturge-Weber syndrome, although not usually manifest as temporal lobe epilepsy, are certainly more commonly seen in children with epilepsy than in adults (5).

The manifestations of temporal lobe epilepsy in children are protean, as indeed they are in all patients, with certain special aspects. Thus stubbornness (52%), temper tantrums (34%), and sleep disturbances (32%) were seen in Glaser's cases (7). Temper tantrums were seen in 36% of another series (12), and sleep disturbance in temporal lobe epileptic children was three times as common as in matched controls (11). Intelligence is generally poorer as measured by I.Q. testing, averaging in the range of 70-80 (7,8), and tends to correlate with likelihood of rage reactions (12). Elements consistent with organic brain impairment include hyperactivity, excitability, poor memory, short attention span and poor visual-motor coordination (7,8).

Behavior varies, "cooperative, good, overcontrolled, but has episodes of impulsive and often violent and destructive behavior" in one study (13); by contrast, "they have a genius for annoying people" (14).

Personality characteristics will be further discussed below.

C. Course

1. Type of Seizures--In Jackson's description of several cases he had consulted on, he noted that sometimes the patient first suffered the "slight fits" or dreamy states but paid no attention to them, believing them of little significance, and only later when convulsions also appeared did their "evil" nature become apparent (15). 63% of Lennox's series of 414 office patients with a history of temporal lobe seizures also gave a history of grand mal seizures, and he noted that they preceded the onset of temporal lobe seizures in 46% of these 250 patients with both types of seizures (3). In a later report he cited a figure of 65% for onset of grand mal prior to temporal lobe seizures, in patients having both (16). 57% of the cases of Currier et al. had grand mal as well as temporal lobe seizures (26). In a study of 300 patients having "psychomotor seizure discharge" (anterior temporal spike focus) on EEG, 66% had both temporal lobe seizures and grand mal seizures; 9% had only grand mal seizures. In this study, Gibbs, et al. also noted that temporal seizures are "rare in children and common in adults" although grand mal are common at all ages (6). Liddell's study of 18 cases of automatism in patients in a mental hospital included five having grand mal in addition, and he noted that "it is usual

in our series for grand mal to have preceded the appearance of psychomotor epilepsy for several years" (17).

Petit mal occurs together with temporal lobe seizures much less often; it was seen in only five of 120 children studied by Glaser (7). Lennox reported a history of petit mal and temporal lobe seizures in 3% of his cases (3). Petit mal is seen primarily in children, whereas temporal lobe epilepsy is more common in adults (see above), so that this low occurrence of both is not surprising. However, there is often difficulty in distinguishing the patients with "petit mal variant" and those with temporal lobe epilepsy clinically; minor motor manifestations (but not the aura) may be common to both, including masticatory attacks and complex confusional automatisms. The EEG helps distinguish these types (5).

It is seen that in the majority of cases of temporal lobe epilepsy other types of seizures may occur, and that since most of these patients are older, the other type is usually grand mal convulsions. I do not think the prior presence of convulsions in a majority of patients having both necessarily implies causality. Many patients never have convulsions, and those who do may have suffered hypoxia, the "common pathway" suggested above, or may have them as the result of secondary generalization from an already

existent temporal lobe focus (5).

2. Control of Seizures---There is general agreement that temporal lobe seizures are difficult to control (18-20), but acceptable control may be achieved in up to 75% of cases (7,20,21). This will be considered below.

3. "Epileptic Deterioration"---In the section on psychological aspects, I shall discuss some of the aspects of intelligence, personality and behavior that have led many to the idea that epileptics worsen in their mental abilities as the course of the epilepsy progresses. Deterioration has been claimed, but in many cases it reflects poor seizure control, organic brain damage also initiating epilepsy, or inadequately treated psychotic states (22). Because of personal or social difficulties, including family problems, inability to take care of themselves, and behavior disorder (17,22), patients may be admitted to hospitals or colonies. Like anyone else spending long periods of time under custodial care, it is not surprising that mentation and behavior deteriorate (22). With interest, concern and adequate control most temporal lobe epileptics should be able to live in society without "deteriorating" and with a modicum of self-respect intact (5,22).

It has also been claimed that deterioration in mental faculties may be due to anticonvulsant-induced folate and/or

vitamin B12 deficiency; I shall also discuss this below. Suffice it to say here that I believe the improvement seen with therapy may at least in part be due to physician interest and concern.

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## Chapter Four

## DIAGNOSIS

### A. Diagnostic Examination

As in all areas of medicine, so in epilepsy the history obtained from patient and family is of preeminent importance. Usually the only knowledge of the seizure the physician has is obtained through careful questioning of patient and family. It is necessary to ascertain all the aspects of the seizure in a descriptive way, in order to properly characterize it. It is also necessary to know the frequency and duration of the seizures and at what times (if any) they seem to occur preferentially. Investigation of cause includes questioning for any noted provocative factors (such as reading or music), for age at onset, for evidence of familial epilepsy, and for evidence of antecedents such as trauma, infection and drugs. Evidence of other neurological damage may be seen, as in school performance or social adjustment.

Physical examination must be careful enough to reveal the presence of "soft" signs, genetic syndromes, evidence of brain damage, vascular malformation, possible intracranial infection, and elevated intracranial pressure.

Laboratory examination derives its appropriateness from the individual clinical situation, but maximally would include determination of serum glucose, urea nitrogen, creatinine, sodium, chloride, potassium, bicarbonate, calcium, phosphorus, magnesium, and more specialized tests in rare metabolic syndromes. Lumbar puncture may be indicated when infection or tumor is suspected, and radiological studies when intracranial vascular malformation, calcification or mass lesion is suspected. (1)

Electroencephalographic examination is of paramount importance in the diagnosis of epilepsy, although there are special problems in the diagnosis of temporal lobe epilepsy, which will be discussed.

B. Electroencephalogram

1. Anterior Temporal Focus---Virtually from the introduction of the EEG to the clinical laboratory, a specific cerebral dysrhythmia was claimed for "psychomotor epilepsy" by the Gibbs'. The initial description, in the late 1930"s, was of a generalized slow wave discharge of 4-6/sec. flat-topped waves of wide amplitude (2,3). In the 1940's Jasper and Lennox and B rody emphasized the deep-lying origin of this abnormality, suggesting it "arises in deep brain structures and is projected to the surface" (3). In their experience the rhythm could be found in cases of lesions in basal ganglia, thalamus, hypothalamus and even cerebellum (2). In this same period, Gibbs pointed out that spikes may also be seen, and when seen, generally had a positive deflection in all leads except the temporal, where it was negative-going (4). Thus he spoke of "fast spikes" and "slow spikes" ("wide, blunt or saw-toothed waves") (4).

As mentioned above, when the active ear electrode on the ipsilateral side was disconnected, and especially when recording was done during natural or induced sleep, all of 300 cases previously identified as "psychomotor discharge" by the criteria above could be shown to have anterior temporal spike foci unilaterally or bilaterally (4). A debate ensued arguing whether the focus itself was in temporal

cortex or deep midline structures, which at least in part led to Gastaut's subdivision of "temporal" epilepsy referred to above (3).

Gibbs, whose position was that an anterior temporal location for the focus could be found in all cases, of course defined the syndrome by the EEG, from which he found his cases. He did admit later that psychomotor seizures (primary automatism) could occur in patients without a "psychomotor seizure discharge" on EEG, reporting cases of this seizure in patients with "classic 3/sec. spike-wave discharge" of subcortical origin. He concluded that these seizures could be set in motion by discharges originating in temporal cortex, frontal cortex or deep subcortical structures (5). He also noted that the anterior temporal lobe focus may accompany grand mal or focal seizures without evidence of psychomotor attacks (4). A recent textbook concurs, in that a specific discharge is stated to be seen in only 69% of cases of temporal lobe epilepsy. The focal activity seen may be spiking, sharp waves, spike-wave, rhythmic slowing, voltage depression, focal delta activity or burst activity (6).

2. "Chasing the Elusive Spike"---A variety of special techniques exist to demonstrate temporal epileptic activity when such is either not apparent on scalp EEG or is not

sufficiently localized to attempt surgery. By far the simplest form of EEG activation is hyperventilation, which probably induces a transient hypoxia in the brain (7,24); photic stimulation, which may induce hypersynchrony, may also reveal epileptic activity, and is also a part of virtually every routine record.

A third benign but more specialized procedure is recording during sleep or after sleep deprivation. Gibbs pointed out the greater likelihood of recording focal epileptic activity during sleep, either natural or induced (4), and other studies concur. Modification of fast activity seen in thiopental-induced sleep may allow specific assignment of a focus in the temporal lobe (7), and pentothal was seen to activate temporal lobe epilepsy in 32% of cases (8). Sleep induced with methohexital activated the EEG in all of 25 temporal lobe epileptics to varying degree, but in none of 25 controls including 10 with a "suspicion" of epilepsy (9), and was claimed to activate only temporal foci, not non-temporal, secondary or dependent foci (10). Intracarotid injection of amylobarbital, used primarily to demonstrate hemispheric speech dominance, has been tried as a technique for suppression of focal epileptic activity, but results are not easily interpretable (7).

Sleep deprivation for 26-28 hours has been used to activate the EEG, and indeed did so in about a third of cases with normal or borderline waking tracings, and in between half and two-thirds of cases with an abnormal waking tracing (11), but others do not find this technique to be of great value (7).

Long-term recording with radiotelemetry has been used to assess interictal activity in waking and natural sleep. Periods of spike suppression were seen to be associated with rapid-eye-movement (REM) sleep, and temporal lobe seizures occurred in intervals of spike suppression (12,13).

Special electrodes include those placed at the base of the skull, via nasopharyngeal placement or transnasal sphenoidal placement, which may be done bilaterally. By themselves or in combination with induced sleep, they demonstrate focal epileptic activity in a large number of cases in which scalp recording is not revealing. Thus scalp alone was sufficient in about one third, nasopharyngeal lead in addition was needed in another third, and sphenoidal lead was necessary in another third to demonstrate a temporal lobe focus (7,14). This is considered so reliable that using these electrodes "failure (of focal temporal activity) to appear in one or two repeated records raises serious doubts about the original diagnosis" (7).

A technique that ought to be mentioned is intravenous Metrazol, which will induce convulsions in virtually anyone. It has also been used to activate the EEG, but the number of false positives limits its use (6).

More invasive techniques include both electrocorticography and depth EEG recording using intracerebral electrodes. Using the former, Gastaut was able to localize temporal discharges to the lateral surface in 24%, to the structures in inferomedian areas of the lobe in 47%, and simultaneously in both (anterior temporal predominance) in 29% of cases (3). These techniques have received extensive use as research tools, such as that reviewed in Chapter Two, and in the delineation of focus of onset in epileptics undergoing surgery for their epilepsy (7). Caution in their interpretation is necessitated for the reason described above: the propagation of afterdischarges into adjacent structures as a possible cause for the manifestations seen (7).

Of interest is the simultaneous recording of surface and depth activity on EEG. A case is presented by Schmidt and Wilder of a patient with intractable temporal lobe seizures and absence of "epileptiform potentials" on scalp and sphenoidal recording, but simultaneous recording with intracerebral electrodes revealed high amplitude spiking in the hypothalamus (1). Lichtenstein et al. reported cases

in which bursts were seen in the temporal lobe not accompanied by clinical phenomena on some occasions, and merging into the patient's characteristic fit on other occasions. They emphasized the variability of the relationship between depth records and surface tracing, and with the observed clinical phenomena (15). In any event, epileptic activity may be present in deep structures of the temporal lobe, may or may not be transmitted to the surface, and may or may not be manifested as seizure activity. This is of great importance in considering the question of interictal psychiatric difficulties, as shall be done below.

c3. Electrographic Variants---Four syndromes have been named having in common visceral and autonomic symptomatology, and the actual significance of all of them is not clear. Chao et al. identified children with autonomic dysfunction as a paroxysmal event, which they called "convulsive equivalents" and related it to an EEG abnormality defined by Gibbs and Gibbs, that of 14/sec. and 6/sec. positive spike activity occurring in bursts during light sleep (16,17). They found that about 40% of those with the clinical syndrome did not have the EEG abnormality, and half of those with the EEG abnormality did not have the clinical syndrome (16). A somewhat similar syndrome was defined by Sheeby et al. as the occurrence of paroxysmal pain in the abdomen and/or

nausea and vomiting, along with other autonomic symptoms; 58% had abnormal EEGs, of which the most common pattern was the 14 and 6/sec. positive spike of drowsiness (18). In the Gibbs' original description they noted that this EEG abnormality was accompanied by syncope, pain, numbness and paresthesias, and vegetative symptoms in a varying percentage of cases, as well as with convulsions in half and with rage behavior in one-fifth (17). All investigators have noted the preponderance in children and adolescents (16-18). Gibbs noted the bilateral, diffuse and independent appearance of this discharge, and hypothesized a centrencephalic origin, naming the syndrome "thalamic and hypothalamic epilepsy" (17). The similarity to Gastaut's diencephalic variety of psychomotor epilepsy will be recalled (3).

Gibbs et al. later defined a different EEG variant of rhythmic theta activity occurring in short bursts lasting one to ten seconds and most clearly seen in the midtemporal area (19). Although, like the 14 and 6/sec. positive spike, it was not temporally associated with seizure activity, there was a highly significant occurrence of the same symptomatology as that described above: headache, dizziness, nausea, vomiting, stomach ache and paresthesias, but no other autonomic symptoms in the usual cases (although these were seen in "atypical" attacks, 13%). A third had

clinical epilepsy, of whom 24% had grand mal and 7% psychomotor seizures (19). They further called attention to the similarity with 14 and 6/sec. positive spiking, in that both were seen during light sleep, were unassociated in time with seizures, and were associated with sensory, emotional and vegetative symptoms; they noted that midtemporal slowing occurred in an older age group (late adolescence), and was more localized than 14 and 6/sec. spiking (19).

Subsequent work has focused on the clinical significance of the 14 and 6/sec. spiking phenomenon. Uncontrolled studies showed a high frequency of behavior disturbance and autonomic dysfunction (20), although follow-up and controlled studies were forced to conclude that there was no specific emotional abnormality to account for the supposed aggressive and criminal behavior (21,22). A recent review emphasizes the inability of controlled studies to demonstrate a consistent relationship of the EEG finding to aggressive behavior or delingency (23). When first described, the occurrence was set at 6%, but Lombroso is cited to have found it in as many as 58% of asymptomatic people as well (23), and this evident non-specificity renders its value doubtful.

A reasonable summary of current opinion would be that the finding of mid-temporal slowing as described is indeed an abnormality of unclear significance, whereas the finding

of 14 and 6/sec. positive spiking during light sleep (especially on "unipolar" electrodes) is of no clinical significance, but a normal pattern seen especially in male children and adolescents (7,23,24). True autonomic seizures probably exist but have no electroencephalographic correlate yet defined (6). The relationship of these phenomena to temporal lobe epilepsy is undetermined when they occur by themselves.

C. Diagnosis

In a provocative essay entitled, "'It', or the ghost in the temporal lobe," Taylor considered the problem of diagnosis thus:

> What then is temporal lobe epilepsy? Is it a psychomotor seizure? Is it an EEG interpreted as showing statistically abnormal features over the temporal lobe? Is it a macroscopic or microscopic lesion in the temporal lobe? Does it have a usual age of onset, affect the sexes differently, prefer one side of the brain? Does it last a lifetime, go away on its own, get chased away by drugs? Does it alter life? (25)

This is a somewhat perverse way of saying that there are really no firm answers to these questions, and the criteria for diagnosis vary with the physician. As I have stated repeatedly, there are no adequate anatomical-clinical correlations to base a definition as a disease, and temporal lobe epilepsy is perhaps best considered a process. The most inclusive criterion would be the presence of a temporal lobe seizure, with or without other types. An EEG abnormality is clearly not enough by itself to diagnose epilepsy, but a more restrictive criterion would be the combination of temporal lobe seizure and temporal EEG focus of abnormality. In order to adequately diagnose the process by this criterion it would be necessary to "chase the elusive spike" as described above, with no certainty of finding it or knowing its significance. Thus I favor a clinical definition,

temporal lobe epileptics being those with temporal lobe seizures, and the EEG being an aid to diagnosis and progress of therapy.

D. Differential Diagnosis

The problem of differential diagnosis is a complex one, due to the protean symptomatology. Without discussing it in detail, I would present the following outline, abstracted from Lennox (26).

1. Psychomotor symptoms
   A. Hypertonus, adversion, masticatory attacks:
      Differentiate mild grand mal, petit mal variant
   B. Excessive activity, mania, fugues, running, bad behavior:
      Differentiate hysteria, emotionality, manic-depressive illness, psychopathy, criminality, "bad boy" (problem child)
   C. Hypotonus, stupor, trance-like state:
      Differentiate petit mal
2. Automatisms
   A. Awareness impaired:
      Differentiate intoxication, metabolic disorder, psychosis, petit mal "status"
   B. Amnesia with intact awareness:
      Differentiate excitement, hysteria, postictal retrograde amnesia, concussion, fever, organic amnesia
3. Psychosensory symptoms
   A. Illusory phenomena (dreamy state, unreality, etc.):
      Differentiate normality, neurosis, hysteria, psychosis
   B. Hallucinatory phenomena:
      Differentiate sensory deprivation, psychosis

Differentiation is usually possible with a careful diagnostic examination, including history, physical and laboratory examination and electroencephalography. Psychiatric consultation may be necessary. Even so, neurologists cite cases (27) and I know of cases where the diagnosis is still uncertain, and the problem becomes one of treatment.

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# Chapter Five

## TREATMENT

### A. Anticonvulsant Therapy

1. Principles of Treatment--The following list of guidelines represent a summary of several opinions (1-3):

    1. Treatment should be begun after the diagnosis is made, as soon as possible.
    2. Where the diagnosis is uncertain, the decision to treat must rest on a relative assessment of individual risks and advantages.
    3. The drug selected reflects the type of seizure to be treated and the toxicity of the drug.
    4. The dose varies with the patient (see below).
    5. Treatment starts with one drug in moderate dosage, which is increased until adequate control is achieved.
    6. If toxicity supervenes and control is inadequate, dosage is decreased below toxicity and another anticonvulsant added to the regimen.

7. Routine follow-up, including monitoring of drug effects periodically by the laboratory (serum drug levels and potential toxic effects), is essential.

8. Social and psychological problems are not left untreated by exclusive concern with the anti-convulsive therapy.

The problems of diagnosis have been discussed, and the drugs that are used will be considered below. Some recent work using serum drug concentrations has delineated variations in response of patients to drugs that can be characterized as follows: serum level of drugs studied (diphenylhydantoin, phenobarbital, primidone) varied directly with dosage; within a given dose range, a wide variation in serum level was seen; the more frequent the seizures, the higher the dose and the higher the serum level; women tended to receive a larger dose, per body weight, than men, and to have a lower serum drug level than men, and to have more seizures; findings of psychomotor slowing, intellectual deterioration, personality change and psychiatric illness correlated with higher levels of diphenylhydantoin and phenobarbital, with duration of seizures, with duration of therapy, but not with frequency of seizures (all correlations statistically significant)

(4,5). The significance of the mental findings is unclear, due to the absence of other possibly significant data on the origin of the findings seen.

2. Anticonvulsants--The patient with temporal lobe epilepsy is best started on either diphenylhydantoin or primidone, but will usually require addition of the other drug (2,3). An alternative statement is the initiation of treatment with primidone and subsequent addition of methsuximide (Celontin), diphenylhydantoin or trimethadione (Tridione) (6). Addition of ethosuximide (Zarontin) may benefit some patients (2,3). Phenobarbital is generally found to be effective, although it is felt by some to worsen the condition (3). A drug said to be effective against most seizures (2), but because of toxicity usually limited to resistant cases (3,6,9), is phenacemide (Phenurone). The most significant side effect is the worsening of behavior disorder or precipitation of psychosis (3,6), a fact noted by Gibbs twenty years ago (7), and a problem also seen with the related drug pheneturide (Trinuride) (8.9).

The toxic and side effects are many, and I list only the major ones here:

| Drug | Idiosyncratic Effects | Dose-Related Effects |
|---|---|---|
| Diphenylhydantoin | Lupoid reaction, blood dyscrasia, hepatitis | Gingival hypertrophy, hirsutism, anemia, ataxia, dysarthria, nystagmus, lethargy, seizures, psychotic reaction |
| Primidone (Phenobarbital similar) | Edema, morbilliform rash, leukopenia, anemia | Drowsiness, nausea, dizziness, sedation, psychotic reaction |
| Trimethadione | Drowsiness, hemeralopia, rash, nausea, marrow depression, nephrosis, hepatitis, lupoid reaction | Sedation, seizures |
| Ethosuximide | GI upset, dizziness, skin rash, marrow failure, lupoid reaction | Sedation, psychotic reaction |
| Methsuximide | Dizziness, nausea, anorexia, "dream-like state," fever, rash, renal dysfunction | |

These effects are seen variably but must be checked for routinely in the course of therapy (3,9,10).

With diphenylhydantoin, primidone and phenobarbital a macrocytic or megaloblastic anemia may be seen in addition as a side effect, and is discussed next. The implications for the patient's mental state are of particular interest.

3. Megaloblastic Anemia--A recent extensive review of the megaloblastic anemias stated that over 90 cases of this anemia in patients on anticonvulsants had been reported, but "many more have not reached the medical literature" (11). Further, epileptics on anticonvulsants may not have anemia but show effects of disturbed folate metabolism:

| Study | Low serum folate (per cent) | macrocytosis (per cent) | megaloblastic marrow (per cent) |
|---|---|---|---|
| Hawkins and Meynell (12) | | 17 | |
| Klipstein (13) | 59 | 49 | |
| Malpas et al. (14) | 31 | 9 | |
| Reynolds et al. (15) | 76 | 9 | 38 |
| Ibbotson et al. (in 11) | 51 | 36 | |

The symptoms, when present, include anemia, weakness, pallor and sore tongue, as well as the mental symptoms to be discussed (11). When anemia is present, folate clearance may be abnormally fast (11), but urinary excretion of formiminoglutamic acid (Figlu) after histidine loading may be normal (16), which has been interpreted as possible evidence for a specific action on limited aspects of folate metabolism (11). If vitamin $B_{12}$ metabolism is not disturbed, all symptoms and signs disappear with folate supplementation (11).

Some patients have megaloblastic anemia and abnormally low serum level of vitamin $B_{12}$; the marrow may be normoblastic, and intestinal absorption of vitamin $B_{12}$ is almost always normal (11). The non-anemic patients with megaloblastic marrows reported by Reynolds et al. had serum levels of vitamin $B_{12}$ averaging 288 pg./ml. (15). Vitamin $B_{12}$ supplementation induced a response in 14 of 26 anemic patients treated only with this vitamin (not folate), although folate alone or both vitamins induced a response in all anemic patients so treated (11). Thus the role of vitamin $B_{12}$ in this anemia of anticonvulsant therapy is anything but clear.

On this background of inadequately understood pathogenesis, attributed by some to intestinal malabsorption (16,17), by others to biochemical effects, which latter is much disputed (11,12), Reynolds has called attention to what he claims is an effect on mental status (15). He found that when on folate supplementation, patients who previously were slowed and blunted in intellectual functions became more alert with increased speed of thought and action, and increased drive, interest, energy, confidence and sociability. He also reports six cases of psychiatric illness in four megaloblastic and two normoblastic epileptics, three with schizophrenia and three with retarded

depression, and although noting that assessment was complicated by the psychiatric therapies employed (drugs, shock), makes no comment about their response to folate or vitamin $B_{12}$, or indeed whether such therapy was attempted (15). Five case reports (18,19) followed, and one study finding mental improvement in 22 of 26 epileptics with low folate (20), but all these results were criticized as being inadequately controlled, with no information about other parameters that might influence the low folate and the response to folate supplementation, and calling conclusions about relationship to mental state "premature" (21). Other reports lined up on the side of a relationship (22-24), and one report comparing epileptics with mental illness and epileptics without mental illness found significantly more patients with low folate in the mental illness group (25). Others lined up against the idea of a relationship (26,27), and three double-blind controlled studies of folate supplementation in patients with low folate, and in one such study also mentally deteriorated (28), found no evidence of any relationship between folate therapy and improvement in behavior, personality, mental state or cognitive function (28-30). Another study of similar design using formal psychological testing has found no significant change after folate supplementation (31).

Thus the situation is quite confusing. The following conclusions appear valid:

1. Evidence of folate deficiency of varying degree is not uncommon among epileptics on anticonvulsant therapy.
2. The mechanism of action of anticonvulsants in this deficiency is unknown.
3. Vitamin $B_{12}$ may be involved in the process, in a way that is not understood.
4. At the present time, there is no good evidence for a relation between this situation and mental state.

The evidence against a relationship is not complete, either. What is the role of vitamin $B_{12}$ (normal in all the double-blind studies)? Are the improvements seen by Reynolds due mostly to attention and concern; are they non-specific effects on mood either due to the vitamins or due to a placebo effect? Does it make a difference in response to therapy whether the patient has had anticonvulsant therapy for years, has had mental illness or deterioration for years, or has had epilepsy for years? A properly designed study to answer these questions would be formidable, if not impossible with the present level of knowledge. A possible conclusion from this review is that folate deficiency

(with or without evidence of vitamin $B_{12}$ deficiency) and mental symptoms should be recognized, considered independent and treated independently.

B. Psychotropic Therapy

In this section I shall discuss therapy with psychotropic agents (antidepressants, phenothiazines) as it affects the epileptic process, realizing that separation of this aspect of their use from the psychiatric is artificial, although convenient.

1. Antidepressants--Animal studies with imipramine (Tofranil), a tricyclic antidepressant, have shown the effect of reducing the severity of Metrazol-induced seizures, as well as evocation of seizure-like EEG patterns by itself (32). Imipramine given to humans also activates the EEG: none of 24 non-epileptics had activation, three of 13 epileptics with non-specific EEG abnormality showed non-specific changes, 11 of 14 epileptics with previous EEG evidence of epilepsy activated this epileptic discharge, and four of six with "epileptic" EEG showed an increase in this activity--a total of 18 of 36 patients showing imipramine activation (33). A further study found evoked or enhanced activity in EEG of 11 of 20 epileptics, 12 of whom were depressed, but only non-specific activity in 18 of 20 non-epileptics, all of whom were depressed, consequent upon administration of intravenous amitriptylene (Elavil), also a tricyclic antidepressant (34). Several cases of grand mal convulsions in non-epileptics on normal doses of tricyclics have been reported (35,36), and a review of massive ingestion of these drugs

in children found that 16 of 25 taking large doses of imipramine had convulsions, often continuous, and 2 of 8 taking amitriptylene had convulsions (37).

2. Phenothiazines--Chlorpromazine has been known for years to be an activating agent for the EEG, provoking focal epileptic patterns and spikes in many epileptics (38-40); it is known to lower seizure threshhold (41) and may increase seizure frequency (40). On the other hand, several reports emphasize its effectiveness in epilepsy: a "good clinical response" in 26 of 31 temporal lobe epileptics (42), marked reduction in seizures in 2 of 6 epileptics (43), and improvement of behavior in three-fourths of "disturbed" epileptics as well as reduction in seizures in one-fourth (44). Bonafede comments in this report that "it seems plausible to assume that the reduction was associated with a diminution in tension and anxiety, as chlorpromazine is not known to have any anticonvulsive property" (44). This finding of reduced seizures was also seen in 64 of 100 hospitalized epileptics when thioridazine (Mellaril) was added to the anticonvulsant regimen, and ascribed to the same reduction of emotional disturbance (45). In 14 cases of temporal lobe epilepsy and "a wide variety of behavior disturbances" admitted and observed while on anticonvulsant and fluphenazine (Prolixin) therapy, Detre and Feldman found no case of

increase in seizure frequency. In some, after increasing anticonvulsants had failed, the phenothiazine succeeded in controlling the seizures; in others, psychotic behavior disappeared with adequate anticonvulsants alone (46). Detre later wrote, "Some patients respond best to anti-convulsants alone, some to psychotropic agents alone; others require a carefully chosen combination of the two." (47)

C. Surgery

The criteria for determining when and in whom removal of the temporal lobe will be of benefit include the following:

1. Evidence of a focal lesion---
   A. Clinically, in the seizure pattern;
   B. Electrographically, the focus on EEG corresponding;
   C. A pathological lesion seen at surgery corresponding to all other focal evidence.
2. Relative stability of the focus.
3. Mental and social state to reap the greatest benefit from potential improvement.
4. Standard criteria of "good surgical risk".
5. Total failure of all other forms of therapy, including medical and psychiatric.

These guidelines (3,48) are points of general agreement, although their application in a given patient may be a source of considerable contention.

Penfield, one of the pioneers in this field, reported in 1954 the results of ten years of surgery for temporal lobe epilepsy, using the technique of removal of deep structures as well as cortex (49): in 51 cases, 14 were cured, 13 had rare attacks, 13 had "worthwhile" results,

and only 11 were unchanged or worse; 16 others were lost to follow-up, and there had been one death (50). In 1964, Falconer published the results of 100 cases he had operated on: 43 of 47 with mesial temporal sclerosis were improved, 28 seizure free; 19 of 21 with small hamartomas were improved; 7 of 10 with small scars or "infarcts" were improved; and 14 of 22 with equivocal lesions were improved by surgery (51). It is interesting that Polish and Russian surgeons report successes of the same magnitude, 70-75% improved (52,53). In these reports, when the focus is unilateral, good results are to be expected in 65-75% of cases. However, when the focus is bilateral, a good result may be obtained in only 21% (54); in another report, 21 of 33 with unilateral foci had successful surgery, but only 7 of 29 with bilateral foci were successful, and of these, when the foci showed no predominance to either side, only one in 11 had successful results (55).

An alternative approach to removal of the entire lobe (to the vein of Labbe, including amygdala and anterior hippocampus) has recently been stereotaxic destruction of the electrographic focus; this has produced a good surgical result (fewer seizures) in from 50% (56) to 70% (57), although some have had to go on to lobectomy. The lack of cognitive deficit after surgery is stressed (57).

Personality and psychological changes after temporal lobectomy were studied by Hill et al. in 1957 in 27 patients followed for 2-5 years. When the dominant lobe was removed, 19 showed poorer auditory learning ability; aggressivity was affected, reduced in 12 and "turned in" or associated with depression in 11. The sexuality of 16 cases was studied, and found to be more involved and satisfying in 13; one was impotent and one perverse (58). Falconer later wrote that when seizures improved, so did a psychiatric disorder (59), but this is an oversimplification. James studied 68 cases, all with normal intelligence, 65% with marked and 25% with moderate personality disorder that correlated with the severity of the process. 57% of these cases were improved in psychiatric status after 1-6 years follow-up (60). The problem of psychosis has been studied independently; 4 of 7 psychotics among James' cases recovered or were improved (60), and when Serafetinedes studied psychotics according to form of psychosis, he found a difference in result. 12 of 100 cases operated on were psychotic; all were followed 1-9 years. The results are given in Table 1.

Table 1

| Sex | Acute Confusional Psychosis | | | Confusional Psychosis & Paranoid/Aff. Sx. | | | Paranoid & Affective Sx. | | | Schizophrenic like Psychosis | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Cure | Impr. | Unch. | Cure | Impr. | Unch. | Cure | Impr. | Unch. | Cure | Impr. | Unch. |
| Male | 2 | 0 | 0 | 0 | 2 | 0 | 1 | 3 | 0 | 0 | 0 | 0 |
| Female | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 2 | 0 | 1 | 2 |
| Total | 2 | 0 | 0 | 0 | 3 | 0 | 1 | 4 | 2 | 0 | 1 | 2 |

He concluded that the acute confusional episodes, which had always been post-ictal, were directly related to epilepsy, whereas the cases with paranoid and affective symptoms were inconstantly associated with improvement in seizures. The three cases of schizophrenic-like psychoses, all young girls, responded poorly (61).

The improvement in intractable epilepsy, the low rate of complications, and the relatively small mental damage with a chance of improvement in mental state lead to the conclusion that, in properly selected patients, temporal lobectomy is a beneficial form of therapy for temporal lobe epilepsy.

The special problems of patients having good results have been investigated from a psychosocial point of view by Horowitz et al. In patients undergoing stereotaxic surgery, patients unrelieved of seizures showed significant mild worsening of psychosocial function, and patients relieved of seizures did not improve significantly (62). Looking at the reasons for this, he identified six types of personality: adequate, retarded development due to epilepsy, inadequate, inadequate due to epilepsy, organic brain syndrome, and decompensated. Non-improvement could be traced to cognitive impairment of organic brain syndrome type, pathological responses to impairments and side-effects of surgery, and to failures in personality development,

depression and/or paranoia. The cognitive deficits included memory impairment, perseveration, sequencing error, defective categorization and set preservation, confabulation and poor abstracting ability; reactions to these included shame, frustration, irritation and depression (63). The issues of psychosocial rehabilitation, then, could be described as intrapersonal and interpersonal aspects of excessive expectation, withdrawn dependency, revision of self-image and generation of a new identity (62).

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## Chapter Six

## PSYCHOLOGICAL ASPECTS

### A. Social Attitudes

1. Marriage--Laws forbidding the marriage of epileptics have only come under attack within the last 25 years. The 1757 Swedish law was objected to by Ahlström, who found in an investigation of 897 epileptics a prevalence of 1.5% among their relatives, a marriage rate similar to the general population, and a reduced fertility; epileptics with mental and emotional disturbance were significantly more likely to be unmarried (1). A 1587 Iceland law forbids marriage among epileptics, and allows annulment on the grounds of epilepsy, but this "is hardly so strict to apply to any but those with severe epilepsy" (2). A Nazi law in Germany in 1936 not only prohibited marriage but required sterilization; laws prohibiting marriage have been in effect in all of the United States at some time, but many are poorly enforced or repealed (2,3).

Several studies have shown a reduced marriage rate among epileptics, besides that of Ahlström. Pond et al. found that among 245 epileptics in general practices serving

39,500 people, in the age group 21-59 years old 62% were married, and single young men were in an excess over the general population (4). Gudmundsson found that among 987 epileptics, those over 18 years old were married in 67% of cases for men and 60% of cases for women (2). A much more restricted study of 79 epileptics referred for lobectomy for temporal lobe epilepsy found 73% unmarried in the age range 21-59 years old, compared to 21% in the general population (5). There is a reasonable agreement among these studies that marriage occurs less often among epileptics having frequent seizures, and less often among men than women with epilepsy.

2. Employment--Dennerll cites studies showing employment rates among epileptics from 74-90% (6). In temporal lobe epilepsy, Jensen's 79 patients for lobectomy were employed in only 43% of cases (5); a more representative sample of 666 temporal lobe epileptics found 88% of those of working age to be employed (7). The factors operating to make employment difficult have been felt to be social prejudice, mental deficiency and activity of epilepsy (6). Pond and Bidwell found employment problems in 40% of cases, usually long periods of unemployment or demotions in work. Those with problems predominated in the lower classes, tended to have more seizures, and were more likely to have temporal

lobe epilepsy, mental deficiency or psychological difficulty (8). A small number, 10% in Pond and Bidwell's study, are probably "unemployable" due to these difficulties. Dennerll emphasises that the difficulty in employment may indeed be societal (see below), but also is personal: seizures and employer attitude have a role, but so do lack of social competence, lower intellectual ability, brain dysfunction, work attitudes, motivation and personal grooming and hygiene, operating for epileptics as for anyone else (6).

3. Social Class--A variety of factors probably act to place epileptics as a group in somewhat lower social classes than the general population. Pond et al. found lower social class correlated with greater seizure frequency, with a younger age group and with single men (4); Jensen's survery of temporal lobe epileptics found that the parents tended to be of lower social class, although the patients tended to be of even lower social class (5). Only very carefully designed studies can evaluate this element, as many (64% according to Folsom (9) for Massachusetts) are under private care, tend to have a better social prognosis, and may not come to be studied by university-based investigators. Pond et al. and Gudmundsson, by the breadth of their investigation seem to include this element of the epileptic population, and concur in the finding of a greater preponderance of epileptics in

lower social classes (2,4).

4. Institutionalization--Using very rough estimates, including public data on epileptic colonies and published studies of epileptic groups in mental-defective hospitals and mental institutions, Pond and Bidwell estimated that 10% of the epileptics in Britain were under the care of institutions (8). Folsom quotes a similar figure for this country (9). From one-to three-fourths of these may be temporal lobe epileptics (10-12). An entire issue of Epilepsia was given to the issues of social function, institutions and special centers (13), a general consensus being that every effort should be made to keep epileptics out of institutions, although specialized care may be needed (8,10,13). Factors that operate to place epileptics in institutions include mental deficiency, severe behavior disorder, inability to support oneself, and unwillingness of the patient's family to provide necessary care (10,14).

5. Epilepsy and the Law--The question of epilepsy being associated with crime is an old one, noted a century ago and restated in 1965, and identified as a source of the notable social prejudice under which epileptics suffer (15). It has been claimed that epileptics commit "terrible" crimes as a result of the process in their brain, and thus do so in excess of the general population. One study of EEG in

criminals found significant epileptic activity, but the cases had been referred because of the possibility of organic disease (10). Similar studies of behavior in institutionalized epileptics are invalid for the same reason. More acceptable summaries come from the large surveys; Ahlström found the prevalence of patients over 25 who had committed indictable offenses to be similar to that of the general population if patients with mental deficiency were excluded (who had a higher rate of criminal behavior) (1). Gudmundsson found three times as many epileptics to have been convicted of criminal offenses as the general population, but did not account for mental normality or deficiency; half of those with criminal records had personality changes of the "epileptic" type, whereas these changes occur in only one-fourth of all epileptics (2). Thus once again, certain factors such as mental deficiency and behavior disorder in some epileptics account for a commonly held misconception about all epileptics. The danger of selective bias in investigation is also illustrated by these studies and the conclusions drawn from them.

Temporal lobe epilepsy has been believed to be the form most likely to involve criminal behavior, and will be further discussed below.

Laws regulating driving motor vehicles seem more reasonable, and receive more general support. Certainly the wisdom of licensing epileptics is proportional to their expected freedom from having a seizure while driving. The seizure-free interval varies; three years even if on drugs in Britain, two years off all drugs according to the American Medical Association (2). The number of epileptics holding driver's licenses in the large surveys of epileptics range from 15% in England (4), 20% in Denmark (2), to 36% in Iceland (2), varying with the strictness of the national laws, and including some people with frequent seizures. Unfortunately, the single large survey of temporal lobe epileptics did not report such data on driving (7).

6. Prejudice--The data on social adjustment reviewed above to a great degree reflects the substantial prejudice in the mind of the average citizen concerning people with epilepsy. The extent of this can be seen from the following table, derived from a report discussing a nation-wide random sample of opinion in response to the questions, "Wound you object if your child played with an epileptic child?", "Should people with epilepsy be employed?" and "Is epilepsy a form of insanity?" Responses to similar questions put to people in Germany and Britain are also presented. (All values are per cent.)

| Question/ Response | United States 1954 | 1959 | 1964 | 1969 | West Germany 1967 | Britain 1969 |
|---|---|---|---|---|---|---|
| Employed?/Yes | 60 | 75 | 82 | 76 | -- | 57 |
| Play with child?/No | 69 | 67 | 77 | 81 | 63 | 68 |
| Insanity?/No | 68 | 74 | 79 | 81 | 73 | -- |

It can be seen from this data (15) that prejudice, although still great, seems to be lessening, due largely to programs of information undertaken in the last 25 years.

In this same study, Bagley chides physicians who have related epilepsy to a variety of anti-social behavior without evaluating the likelihood of chance association, and seeks to test the hypothesis that prejudice against epileptics is based on each person's fear of losing control of himself. Thinking that spastics would be less likely to arouse this fear, and that racial prejudice shares a similar basis in fear of instinctual strength and primitiveness, he compared responses to these ideas in 574 teenagers, 104 working adults, 211 college students in Britain. Twice as many opposed employing epileptics as spastics, and on a social distance scale epileptics were rejected to the same degree as "colored" races and Jews; indeed, they were rejected significantly more often

than people with mental illness, suggesting that "if epilepsy were seen as a form of mental illness, then acceptance of epileptics might be increased." (15)

These results can only be suggestive of the extent of social prejudice against which epileptics must struggle.

B. Causes of Psychopathology

1. Social--Having surveyed prejudice against epileptics, Bagley concludes:

> The role of neurological factors in behavior disorder seems to be to increase the vulnerability of the child to this disorder, so that children who are brain-damaged and have minor fits and who have had fits for a number of years and who have a poor environment and who have rejecting parents are particularly likely to manifest aggressive behavior disorder. In our statistical analysis the role of the parental attitudes was by far the strongest influence on the behavior disorder. (15)

Pond and Bidwell concur in thinking the role of seizures less important than the reaction of the environment to the person having them--for a child, primarily his parents, school and peers (8). Pond restates this idea elsewhere, and cites a study in which he found the prevalence of broken homes and psychopathy to be the same for children with behavior disorders whether or not they had epilepsy, but much higher than that for epileptics without behavior disorder (10).

The folklore about temporal lobe epilepsy and crime may derive from the behavior associated with the seizure, which although seldom purposeful enough to be "criminal" is frequently unusual enough to be socially unacceptable. During seizures temporal lobe epileptics may undress, scream, urinate, act aggressively---behavior "poorly tolerated by the community"(12) and advanced as the reason so many epileptics in

institutions have this form of epilepsy (11,12). Others enter because of more clearly psychotic behavior (11), to be discussed below.

2. Intelligence--In a review of literature on intelligence testing in epileptics prior to 1953 Folsom concludes that the range of intelligence found, when adequate samples are used, corresponds to a normal distribution (9). Serial retesting did not demonstrate "deterioration" attributable to seizures. However, she notes that several studies have shown a significant difference in epileptics with recognizable brain damage; inclusion of this group, who have lower I.Q. scores, will skew a population of epileptics unless this is taken into account. I.Q. was not related to duration of epilepsy, severity or frequency of seizures or absolute number of seizures. It was noted that this did not hold for children, who had lower I.Q. scores than normal and in whom serial retesting did detect deterioration with severity and frequency of seizures. She suggests that interference with intellectual growth may occur as a result of interference of schooling (a social factor), failure to acquire the skills tapped by the intelligence tests, particularly the Stanford-Binet Test, or major seizures associated with cerebral hypoxia may cause permanent damage to the brain. All may act together, and may be modified by the normality of the environmental

interactions as suggested above (9).

These general conclusions are upheld by the results of more recent work. Gudmundsson's review comes to similar conclusions (2). Pond noted similar factors affecting the intelligence of epileptics, and noted in addition the effect of the type of seizure. He had thought that the lowest mean scores occurred in patients with grand mal convulsions, the next highest scores in those with temporal lobe seizures, and the highest in those with petit mal seizures, temporal lobe epileptics having "nearly average intelligence", but he could not confirm these general impressions. Seeing only psychiatric patients, he found temporal lobe epileptics to have higher scores than all others, a fact he attributed to better sampling, only dull and severely affected epileptics with grand mal or petit mal seizures coming to psychiatric attention (10).

Looking at patients with temporal lobe epilepsy due to tumors, compared to those without tumor, Bingley noted the effect of involvement of the dominant hemisphere, 46% having intellectual deterioration, as opposed to 19% when non-dominant. Mean I.Q. scores for patients with a tumor on the dominant side was 82, for an EEG focus on the dominant side 92, for bilateral EEG foci 90, and for either tumor or EEG focus on the non-dominant side 106 (16). This illustrates

the effect of brain lesions and the interaction with cerebral dominance.

Studies comparing temporal lobe epileptics against other types of epileptics have not all come to the same conclusions. Quadfasel (neé Folsom) and Pruyser compared 19 cases of patients with grand mal and temporal lobe seizures against 19 cases of patients with only grand mal seizures, all adult:

| Test | Temporal Lobe | Grand Mal |
|---|---|---|
| Full Scale I.Q. | 107.0 | 107.0 |
| Verbal I.Q. (V.I.Q.) | 100.4 | 104.0 |
| Performance I.Q. (P.I.Q.) | 113.8 | 109.4 |
| P.I.Q. minus V.I.Q. | 13.4 | 5.4 |

They conclude that this demonstrates a significant (p .01) impairment in verbal functioning in the temporal lobe group (17). A study of similar design by Mirsky et al. found the following:

| Test | Temporal | Frontal | Centrencephalic |
|---|---|---|---|
| Full Scale I.Q. | 100.6 | 93.9 | 85.7 |
| Verbal I.Q. (V.I.Q.) | 99.5 | 96.4 | 89.6 |
| Performance I.Q. (P.I.Q.) | 101.2 | 91.6 | 83.7 |
| P.I.Q. minus V.I.Q. | | -4.8 | -5.9 |

Thus they were unable to conclude that there was a significant difference in verbal functioning (18). Later work by Small et al. also found no significant difference in any

of these measures between temporal lobe epileptics and patients with either petit mal or other focal seizures (19). Stevens et al. compared temporal lobe epileptics with "centrencephalic" (petit mal or grand mal) and frontal focal epilepsy, and although not all the scores are reported, found no significant difference in full scale I.Q., but significant differences (temporal lobe epileptics better) in verbal I.Q. compared to frontal focal, and in performance I.Q. compared to centrencephalic epileptics (2). Studies which have reported results for sub-tests of the WAIS indicate that temporal lobe epileptics are significantly better than centrencephalic epileptics in Arithmetic and Comprehension, but differ in relation to scores on other sub-tests and in relation to other focal epileptics (19,20).

Studies of I.Q. in children with temporal lobe epilepsy also find poorer than normal performance, without identifying the reasons for this impairment (21,22).

3. Memory--Folsom states that memory impairment is claimed to be frequent in epileptics, and suggests that the main deficit is in attention. The digit span subtest of the WAIS is given as being low in several studies of epileptics, but without control for the low values found in normals (9). More recent studies that have looked at "memory" WAIS sub-tests, although not controlled against normals, have not

demonstrated significant differences between temporal lobe epileptics and other epileptics (19,20).

Tests using the Wechsler Memory Scale have been somewhat more precise. Quadfasel and Pruyser found Memory Quotients (M.Q.) to be 93.4 for temporal lobe epileptics having both temporal lobe and grand mal seizures, and 102.9 for patients having only grand mal seizures. The difference between Full Scale I.Q. (WAIS) and M.Q. is 13.6 for the temporal lobe group, and is said to indicate memory impairment ($p<.02$) compared to the other group (17). Mirsky et al. could only identify a similar trend (not actually significant) for this measure in their data, particularly when the EEG foci were bilateral (18). Stevens et al. found significant differences on certain aspects of the M.Q. when compared to "centrencephalic" but not to other focal epileptics (20. Quadfasel and Pruyser further attempted to identify verbal memory and non-verbal memory, and on these measures their temporal lobe group was significantly worse in the verbal area than their grand mal group. Bearing in mind their finding of impaired V.I.Q. (see above), they suggest that the observed impairment in memory is largely due to a general impairment in verbal functioning (17).

Finally, Folsom's claim that the memory disturbance is a difficulty in attention is not supported by the finding

of Mirsky et al. that results of a test (continuous performance test) claimed to measure attention show significantly less impairment in temporal lobe epileptics than in patients with centrencephalic epilepsy (18). It is difficult to draw meaningful conclusions from this data. The variation in composition of the test groups between the studies reviewed here probably accounts for some of the lack of concurrence. It is not clear whether epileptic involvement of the temporal lobe and limbic system accounts for the disturbance in memory, as might be suggested by the data considered above under "Functions."

4. Tumors--That tumors in the temporal lobe may cause psychopathology as well as seizures has been alluded to above, although the two effects have not always been separated. Gal identified 61 cases of temporal lobe tumor of whom mental symptoms were the presenting symptoms in 37. 50 of the 61 had mental symptoms, including an organic syndrome (drowsiness, blunted sensorium, thinking and attention, lack of interest and apathy) in 30, mood disorder in 9 (three depressed, two anxious, four euphoric), paranoid psychosis in one and a personality change in one. Ten also had "parietal" symptoms (23). Malamud collected several cases of tumor in the limbic structures, poorly documented as to the types of seizures, if any, that accompanied them. Nine cases of

temporal lobe tumor presented with psychiatric disorder: schizophrenia in four, depression in four and anxiety in one, Two cases of tumor in cingulate gyrus presented as schizophrenia, and four tumors of the third ventricle presented as schizophrenia (24). Bingley found 67 cases of temporal lobe tumor without papilledema, of whom 22 showed intellectual deterioration, 9 emotional change, and 36 slight or no change. The changes seen were in intelligence (see above), attention and concentration, and those said to be characteristic of the "epileptic personality" "discussed below) (16).

5. Temporal Lobe Electric Activity--As mentioned above, epileptic activity in the temporal lobe may or may not be transmitted to cortex and may or may not be associated with clinically evident seizure activity. In fact, Lichtenstein et al. found the great majority of EEG bursts unassociated with overt clinical phenomena (25). However, when Kooi and Horey gave psychological tests to epileptics as the EEG was monitored, "disturbances in higher integrative mental processes turned out to be significantly associated with paroxysmal cerebral activity." The disturbances consisted of non-response, "I don't know," or inappropriate responses, all of which also occurred in the absence of EEG bursts (26). Stevens et al. noted a relationship between periods of spike suppression

and subsequent temporal lobe seizures, suggesting a "discharge" phenomenon (27); this idea forms the basis of a hypothesis of Dongier concerning the longer affective disturbances with normal consciousness seen in temporal lobe epileptics, and often terminating in a seizure. Perilesional spike activity would generate the mood swings, and cortical desynchronization would account for the "normalization" or spike suppression in the scalp EEG. Presumably the effect of building up to a release in a seizure would be similar in nature to that of electroconvulsive therapy (28). In fact, Ervin et al.'s first case would induce a convulsion by omitting medication when mounting anxiety or depression became unbearable (29).

Goldensohn and Gold reported five cases in which disturbed behavior lasting up to 72 hours was associated with generalized epileptic activity in the EEG (30), demonstrating that it could be "ictal" as well as "pre-ictal." When Brady attempted to find a relationship between increased seizures and increased behavior disturbance, no significant relation emerged for the 20 hospitalized temporal lobe epileptics studied. However, there was a trend to this relation, and the sample size may have been too small (31).

Ervin et al. found 42 patients with interictal temporal spikes, 31 of whom had epilepsy, 16 with temporal lobe and grand mal seizures, 12 with only temporal lobe seizures.

24 of the 28 patients with temporal lobe seizures were classified schizophrenic, and 9 of the 11 with no seizures were classified schizophrenic. 20 of the cases with no previous psychiatric history were no less impaired on psychiatric testing than the others with such a history (29). This study would suggest that temporal lobe spike activity is associated with psychiatric disturbance, but is weakened by the large bias in sampling (psychiatric referrals), the small sample size, and the use of Rorschach tests without appropriate controls in measuring disturbance of mental processes.

6. Folate--As discussed in detail above, the claims made by Reynolds and his co-workers that folate depletion or deficiency, interacting in some cases with abnormal metabolism of vitamin $B_{12}$, was responsible for mental deterioration, mental illness and "bradypsychia" have not been confirmed by the results of several controlled studies. It is sufficient here to mention that this is another hypothetical cause of psychopathology in epilepsy, largely unsubstantiated, but possibly interacting with other causes such as heredity, brain damage and psychosocial maladjustment. It is interesting to note that sometimes the seizures get worse as the mental state improves on folate and vitamin $B_{12}$ supplementation, which is remotely in accord with the data reviewed above.

Reynolds has summarized much of his thinking on the role of these vitamins in the biochemistry of epilepsy and schizophrenia and their common relationship of antagonism (first proposed by Meduna) (32), but the idea has not been generally accepted.

7. Psychodynamics--In 1963 (33) and in 1964 (34) Glaser reported studies on temporal lobe epileptics which found the presence of cerebral defects felt to predispose to the development of psychosis. Of 37 such patients, half had defects in memory, attention and time sequencing, consistent with the data reviewed above (34). However, the striking finding was the presence of fluidity of thought processes, manifested during psychological testing as loss of a train of association, or as word-finding difficulties or "tracking" difficulties. He also noted fluctuating levels of alertness and of accuracy of perception. Associations tended to be loose and response "concrete" but without evidence of bizarre content or mode of thought, and without signs of withdrawal from reality (33,34). Unfortunately, these findings are difficult to reproduce by other examiners, as they are not standardized or quantitative, and have not been found in subsequent studies of temporal lobe epilepsy by Small et al. (35); Flor-Henry's study of psychosis in temporal lobe epilepsy does not report their presence or absence (36). This will be

discussed further below, with psychosis.

The "misinterpretation" of sensory data in the temporal lobe seizure has been put forth as an explanation for the observed psychopathology. Gibbs thought that if the cortical centers interpreted experience as unpleasant, a depression might result; if the badness of experience was attributed to a special influence, the result might be paranoia. Bizarre judgment defects might be associated with psychopathy and schizophrenia (37). Williams interpreted the events of the temporal lobe seizure as misinterpretations of the relations of the self to internal and external environment, which might result in disorders of behavior and thought (38). Other theories abound to explain the generation of psychopathology in temporal lobe epilepsy, but they have in common no experimental evidence or scientific basis other than speculation from known data that is incomplete.

Epileptics can use their epilepsy to provoke seizures (through reflex mechanisms, emotions or as yet undefined ways) in certain rewarding situations. The release of emotions has been mentioned (29). Escape from a difficult situation is another such reward (39,40), as are the avoidance of charged material (41,42) and manipulation of others through invalidism and dependence (39,40). These do not cause the observed psychopathology so much as reflect it, in an interaction with

the social environment that is disturbed or in the failure to resolve significant developmental issues.

Some highly fanciful and completely non-scientific explanations have been given, based on psychoanalysis of epileptics. Revitch cites one such idea, that the seizure imitates joy in dying, and in it "the epileptic experiences birth, death, and sometimes rebirth. The epileptic patient flees the dangers of life into a temporary death, then to be reborn" (43). Aggernaes thought seizures, particularly complex ones, to indicate a weak personality structure (44). Epstein and Ervin saw the seizure as a form of denial (45), but Rodin et al. saw it as a failure of repression (46); Ostow has stated this theory thus (47).

> Some kinds and distributions of temporal lobe disease may confer pathological overactivity upon one or more unconscious fantasies so as to preclude adequate repression, or they may interfere with the repressive mechanism itself. In either case, the consequence will be imperfect repression of an unconscious fantasy and, according to psychoanalytic theory, this should give rise to neurosis or psychosis.

Little light is shed by this sort of untestable hypothesis.

A more fruitful result stemming from the correlation of seizures, dreams and electrical stimulation of the brain is that of Ferguson et al. who analyzed five such cases (48). They found "organic mental deficits" in recent memory, time sequencing, auditory receptive ability and critical comparative

ability, manifest as lack of continuity of experience, disorientation, association bias, comprehensive difficulty and fractionation of experience and its incorporation. This is consistent with the data on memory reviewed above, and strongly supports the finding by Glaser of similar deficits in psychotic temporal lobe epileptics. Stevens et al. could not find evidence of these deficits in her studies, as described above (20). However it remains to be seen whether this line of investigation, fraught with methodological difficulty and tendency to hypothesis, will ultimately prove fruitful.

C. Personality Studies

In 1962 Tizard published a critical review of studies of the "epileptic personality," pointing out the existence of at least five general theories:

1. All or most epileptics share a characteristic personality, consisting of perseveration, viscosity and impulsiveness.
2. Traits claimed to be epileptic are found in the general population; there is no specificity.
3. There is no typical personality, but there is a higher incidence of neurosis in epileptics.
4. Epileptics tend to have a personality resembling that of people with organic brain damage.
5. Different personality traits are associated with different types of epilepsy.

This can be taken as a guide to the many studies of personality in epileptics (49).

The characteristic personality seen in epileptics has been claimed to include perseveration; an entity variously called "stickiness," "adhesiveness," "viscosity," all referring to an inability to shift mood with modified

circumstances, with a persistence of the same emotional tone after a change in stimulus (43); explosiveness or impulsiveness, associated with moodiness, temper outbursts, rigidity and pedantry. Other traits that have been suggested include religiosity, meticulousness, suspiciousness and selfishness (2,43,49). Certainly many of these traits are found in the general population, and most of them cannot be measured accurately in studies of personality. Thus when they are reported, it is on the basis of clinical observation, open to extensive observer bias. This personality structure has been called "ixophrenia," with derivative terms "ixoid" and "ixothym" (referring to traits also found in the general population). Using these terms, in a retrospective study of 987 epileptics, Gudmundsson found 48% normal, 7% ixothym, and 27% ixoid, with ixoid people tending to also have mental deficiency (2). In a similar survey of 897 epileptics, Ahlström found symptoms of perseveration and adhesiveness in 7% of cases with unknown etiology, 15% of cases with probably known etiology, and 15% of cases with definitely known etiology, suggesting that they reflect brain damage (1).

A number of studies reviewed by Tizard attempt to place the prevalence of personality disorder in temporal lobe epilepsy at approximately half of cases (49). Reports within the past ten years include the following data (all values in per cent) (50):

| | Guerrant (51) | | Vislie (52) | | Small (19) | |
|---|---|---|---|---|---|---|
| | Temp. | Grand mal | Temp. | Grand mal | Temp. | Grand mal |
| (Number of patients | 32 | 26 | 84 | 58 | 25 | 25 ) |
| Adhesive | 41 | 30 | 8 | 7 | 8 | 20 |
| Slowed | 25 | 12 | 8 | 16 | 8 | 20 |
| Apathetic | 22 | 12 | 16 | 24 | -- | -- |
| Labile mood | 31 | 20 | 38 | 30 | -- | -- |
| Impulsive | 34 | 35 | 13 | 16 | -- | -- |
| Compulsive | 19 | 15 | 16 | 10 | 8 | 0 |
| Anxious | 53 | 46 | 41 | 28 | 12 | 20 |

Unlike many previous studies, the samples compared above do reflect representative populations of epileptics, and the data does not allow the conclusion that temporal lobe epileptics suffer a personality disorder out of proportion to other epileptics or in a majority of cases.

Some methodological considerations are pertinent to the review of epileptic personality. The tools that have been used include observation, self-designed ratings, Rorschach, the Halstead-Reitan test battery, and the MMPI. The first two suffer from observer bias and the first from lack of reproducibility, as I have mentioned. The Halstead-Reitan test battery has specificity in comparing groups with and without brain damage, if the latter are not schizophrenic; however, if the group without brain damage suffers affective illness,

specificity is lost in a comparison with a group of epileptics (53). Thus studies using these tests in epileptics may be distorted by the simultaneous presence of either form of mental illness in test group or in control, as is frequently possible. The Rorschach test results have been extensively criticized by Pruyser (54) and Tizard (49), who point out the following:

1. In developing interpretations of the test, Rorschach tested many epileptics, and himself believed in their having specific personality traits that he could identify.
2. Responses to the test vary with intelligence, which must be (and seldom is) controlled for.
3. Rorschach test results cannot be used as a diagnostic technique, only a descriptive one.

Without taking into account these factors, the published data can be collected to support and to refute each of the five theories outlined by Tizard (49). When these factors are taken into account, the pattern that emerges is that epileptics are indistinguishable from non-epileptics with similar behavioral profiles, and the theory of a specific epileptic personality with identifying traits cannot be supported (49,54).

Studies using the MMPI, a complicated battery of tests completed by the subject and scored by the examiner with the aid of a computer, have not yielded uniform results. Small et al., Mignone et al. and Stevens et al. could find no difference of significance between temporal lobe epileptics and other epileptics (19,20,55), although Stevens et al. suggests that temporal lobe epileptics do better than the others (20). As a group, they score higher on all subtests than do "normals" (college students); Mignone et al. found this true for all subtests except the Masculine-feminine (55); Stevens et al. noted the Schizophrenia scale tended to be the most abnormal in all epileptics (20); and Meier and French found schizoadaptive traits excessive in bilateral as opposed to unilateral focus temporal lobe epileptics (56), which was not confirmed in Mignone et al.'s study (55). Finally, Stevens et al. compared MMPI profiles with psychometric test results (WAIS, WMQ, and Halstead-Reitan), and could not correlate personality indices with specific deficits in psychometric testing (20).

In summary, it may be concluded that there exist personality traits in the general population which seem to occur more often in epileptics. Whether they are those traits which have been claimed (perseveration, viscosity, impulsivity, rigidity), and whether these traits reflect the extent of

organic brain dysfunction associated with epilepsy, have not been demonstrated conclusively. Age of onset, duration, seizure frequency, cerebral laterality and dominance have been associated with psychopathology (see below), but could not be associated with performance on the MMPI (55). Thus the extent to which the causes reviewed above are reflected in personality abnormalities is not clear, and the contribution of these causes as well as that of personality disturbance to the psychopathology of epilepsy is not well delineated. It may be well to recall at this point the heterogeneity of the epileptic process mentioned above, its inconstant association with observable clinical phenomena, and its frequent involvement of parts of the brain at a distance from the site of origin, and invoke the inadequacy of our knowledge of anatomical-clinical correlations in epilepsy as a factor underlying the unclear understanding of observed psychopathology.

D. Affective and Behavioral Disorders

1. Affective Disorders--In Pond and Bidwell's survey of epileptics among 39,500 patients of general practitioners 29% of the epileptics showed some psychological difficulty (8). Of a total of 70 such patients, 34 were neurotic, the rest having behavior problems (children), personality changes (temporal lobe epileptics) and organic symptoms; it is not stated what these neuroses consisted of, but it appears clear from their data that a disturbed environment was associated with at least half of the cases of psychological difficulty. Mulder and Daly studied 100 such patients, all with temporal lobe epilepsy and all presenting with psychiatric complaints. They found that anxiety related to concern over loss of function was prominent in 36 cases, depression thought to be reactive was seen in 16 cases, and schizoid tendencies were noted in 20 cases, all seen (with some overlap of symptoms) in 61 of the 100 patients (57). Among Ervin et al.'s 42 cases of temporal lobe spike foci, 31 of whom were epileptic and 28 temporal lobe epileptic, marked depression was noted in 21, but it is not stated which patients were depressed (29). Ferguson found 16 psychiatric manifestations in 13 temporal lobe epileptics; there were four cases with depressive manifestations, associated with anxiety, paranoia, sociopathy and schizoid adaptation in the

respective four cases (58). Stevens, looking at a clinic population largely indigent, found 28 of 100 consecutive cases of epilepsy to have experienced psychiatric hospitalization; of these, anxiety and mood disturbance (dysphoria) were more common in the temporal lobe epileptics than in cases of grand mal or petit mal epilepsy (50).

Studies of psychosis in epileptics reveal further cases of affective disorder. Studying 536 psychotic episodes in 516 epileptics, Dongier found 153 episodes of depressed affect and 80 of heightened affect, out of 397 cases for which information was sufficient. Although all types of affective disorder were seen in all types of epilepsy, there was a significant trend for affect to be disturbed in psychotic episodes in temporal lobe epileptics as opposed to centrencephalic epileptics, and in particular for a depressive psychotic episode to be associated with temporal lobe epilepsy (28). In a controlled study of 50 temporal lobe epileptics with psychosis, Flor-Henry found that 22% had schizo-affective and 18% a manic-depressive form of psychosis. The manic-depressives were the most "normal" in social and psychosexual adjustment and in intelligence, and tended to have a temporal lobe EEG focus lateralized to the non-dominant hemisphere (36). This finding of laterality in affective psychosis has not been confirmed by others (7,20,55).

Affective disorder, primarily depression, is a commonly stated problem in the clinical experience of physicians treating temporal lobe epileptics, and a large survey of temporal lobe epileptics confirms this impression: of 666 such patients examined, 19% were considered anxious, and in 7 it was felt to be incapaciting; 11% were depressed, including four with severe depression; another 6% were described as having "severe affective disturbance," a total of 36% with affective disorder of varying severity (7).

2. Behavior Disorder--This is perhaps one of the best-known "correlates" of epilepsy, in that it underlies so much of the prejudice reviewed above. Aggression and criminality have been considered above as ictal elements and in relation to causes of psychopathology. Traits of hyperactivity, aggressiveness, destructiveness and violence are seen in instituionalized epileptics, many of whom are also mentally deficient (59,60). It is only necessary here to note that ictal rage does occur, and behavioral disorders including aggressive or destructive behavior may be associated with temporal lobe seizures (30). Also, psychopathy or sociopathy has been observed among epileptics, more commonly in those with mental deficiency (2), and according to Small et al. more commonly in non-temporal than temporal epileptics (19). There may be a trend for behavior disturbance to increase as seizures increase, among

hospitalized epileptics (31). Finally, as described above, ablation or stereotaxic destruction of temporal lobe structures will sometimes result in a reduction of aggressive behavior disorder in temporal lobe epileptics.

3. Sexuality--Although sexual involvement in the temporal lobe seizure is rare, disturbed sexuality is common in the interictal period. Gastaut and Collomb thought global hyposexuality was uniquely associated with temporal lobe epilepsy (61), and the data reviewed above on the marriage rate for epileptics does indicate a significant depression of this index of sexuality (see above). This has been challenged recently; Mignone et al. thought that the sexual hypofunction was not "out of proportion to the overall disorder"(55). Saunders and Rawson studied sexuality in 100 male temporal lobe epileptics, and found 12 cases in which there was a disturbance. Seven were impotent with normal libido, one had "psychogenic" impotence, one had low libido attributed to drugs, one was exhibitionistic, and two had a global hyposexuality (62). Blumer studied temporal lobe epileptics having lobectomy, and found that 29 of 50 were hyposexual preoperatively, and sexuality increased post-operatively if the surgical result was "good", i.e., there was a virtual abolition of seizures (63). Taylor, in a similar study, found only 36 of 100 patients operated on experienced improved libido and

normal sexuality; the others were hyposexual due to ignorance, indifference (older patients), or hesitation. One was hypersexual, and 15 were considered perverse in their manifestation of sexuality (64).

All of this is consistent when it is realized that the marriage rate was lowest for those having the most seizures (those most likely to come for lobectomy), and that sexuality is also influenced by age, maturity of personality, mood and psychosocial adjustment. A patient's sexuality will reflect his position relative to all of these issues (64).

E. Psychosis

Bartlet reviewed the records of Bethlem Royal Hospital and the Maudsley Hospital for epilepsy and psychosis; of 1073 epileptics seen at the hospitals, 12 cases could be found that satisfied his criteria (epilepsy antedating the psychosis, delusions for at least one year), a prevalence of 0.75% which is similar to the general population for schizophrenia. Of the 12, seven of eight schizophrenics and one of three with affective psychosis had temporal lobe epilepsy (65). In Pond and Bidwell's study of epileptics in general practice, none of 245 epileptics showed evidence of psychosis (8). In Ahlström's survey of 897 epileptics, 19 were called psychotic, of whom seven were schizophrenic, the rest having organic dementia; this is a prevalence of 0.78% schizophrenia among epileptics (1).

Mulder and Daly, studying 100 temporal lobe epileptics with psychiatric complaints, found only four with psychosis, of whom two were schizophrenic. 18 others were classified as schizoid (57). In a broad survey of 666 temporal lobe epileptics, Currie et al. found 40 with severe psychiatric problems, of whom 12 were schizophrenic, or 1.8% (7). It may be concluded from this data that temporal lobe epileptics are more likely to have psychosis, and in particular to have schizophrenia, than are epileptics in general. This will be

considered in more detail below.

Pond wrote in 1957 that the problem of psychosis in epilepsy could be considered under five headings:

1. Post-ictal confusional states, with paranoid ideas and hallucinations.
2. Chronic paranoid psychoses with hallucinations in chronic epileptics.
3. Schizoid personalities who have complex auras and hallucinations.
4. Catatonic schizophrenics who have occasional "epileptic" attacks.
5. "Epileptic" phenomena in the EEG of schizophrenics.

Probably only the first two groups are true cases of psychosis in epilepsy. Pond describes the chronic paranoid psychosis thus:

> They include paranoid ideas which may become systematized, ideas of influence, auditory hallucinations often of a menacing quality, and occasional frank thought disorders with neologisms, condensed words and inconsequential sentences. A religiose coloring of the paranoid ideas is common. The affect tends to remain warm and appropriate, which is in contrast to "true" schizophrenia, nor is there typical "schizophrenic" deterioration. (10)

In the first major study of psychosis in epilepsy, Dongier in 1959 analyzed the reports of 536 psychotic episodes in 516 epileptics collaboratively collected from all over Europe. 56% were seen to have acute confusional

episodes, 30% affective disorder, and 10% schizophrenia-like psychosis. 19% lasted less than one day, and 64% lasted less than one week; in 25% of cases the episode was preceded by a seizure, and in 10% of cases it terminated in a seizure. There were 40% with centrencephalic epilepsy, 44% with temporal lobe epilepsy, 6% with other focal epilepsy, and 10% unclassified. Correlations between clinical and EEG features of the episodes on the one hand and the variety of epilepsy on the other were not significant enough to allow one to draw inferences about one aspect from information regarding the other, except for the group as a whole. The centrencephalic group tended to have acute, brief confusional episodes usually beginning with a seizure and lasting several hours. Those with spike and wave patterns in the EEG were felt to represent "petit mal status epilepticus" and those with diffuse delta activity (who were usually adults and whose episode usually began with a seizure) to represent a prolonged post-ictal state. The psychomotor or temporal lobe epilepsy group tended to have longer attacks that seldom began though often ended in a seizure, and disturbance of affect rather than of consciousness was the dominant feature. The EEG in this group was either unchanged or showed increased focal epileptic activity or disappearance of normal and epileptic activity, and was not correlated with clinical features of the attack. These

attacks in temporal lobe epileptics were felt to be most likely a sub-ictal or pre-ictal state (28).

The second major study is that of Stater, Beard and Glithero of the schizophrenia-like psychoses in epilepsy which is widely quoted and probably inaccurate in several respects. They reason that the likelihood of epilepsy and schizophrenia occurring together by chance alone in one individual is the product of their respective prevalence, or (.005) X (.008) = .00004 which is forty per million cases for a population. They further calculate that of these "random" cases, 148 would be living in the area served by their hospital, and that if new cases appeared at a mean age of 29, four to five new cases a year could be expected in the area of their hospitals. They collected 69 such cases from the total intakes of two major clinics for a period of 12 years, which is entirely consistent with the likely chance occurrence of schizophrenia and epilepsy in the population they served, both primarily and as referral hospitals for psychiatric and neurologic patients in Britain. Nonetheless, they concluded that this number of cases "so easily ascertained" represented a "frequency much greater than chance" and thus an association.

The other often-cited conclusion from their study, which the authors themselves later qualified, was that the

schizophrenia occurred significantly after the onset of epilepsy, because the mean age of onset of epilepsy was 14.1 years and the mean age of onset of psychosis was 29.8 years. The correlation coefficient between the two factors was +0.6 and thus highly significant. However, as no cases were found in which schizophrenia preceded epilepsy, this is statistically invalid, and recalculation revealed that the mean age of onset was only significantly different from that expected by chance alone for the females in the study (67), who were only 33 of the 69 cases reported.

Other significant results of their study included the fact that, although as a group all showed delusions of primarily paranoid, religious and unreal nature with ideas of reference, and hallucinations occurred in 52 cases, the thought disorder displayed was of schizophrenic type in 31 and was compatible with organic states in most cases. Flat affect was seen in 28 cases. They conclude that "there is not one of the cardinal symptoms of schizophrenia which has not been exhibited at some time by these patients." The features as described are also in accord with the description given by Pond stated above. With regard to epilepsy, 45 of the 69 cases were temporal lobe epileptics on clinical grounds alone, and an additional 10 cases showed temporal lobe epileptic activity when sphenoidal electrodes

were used in EEG study. There was no significant relation of psychosis to severity of the epilepsy, frequency of seizures or quantity of medication. Etiological considerations, special neurological tests (pneumoencephalography) and psychological testing all revealed a significant amount of organic brain damage. That primary schizophrenics have strong genetic histories of mental disorder is clear, whereas these cases of psychosis in epilepsy had mental illness in relatives at a prevalence no greater than that for the general population, indicating further the secondary nature of the process. Quoting Symonds' idea of an "epileptogenic disorder of function" underlying seizures and mental illness, they conclude that physical (neurological) factors are more important than the psychological theory attributed to Pond in the genesis of the schizophrenia-like psychosis of epilepsy (10,66,68).

In brief but very concise presentation of results of study of 37 patients with temporal lobe epilepsy and psychosis of a chronic, paranoid schizophrenia-like type, Glaser made a number of important observations. The idea that certain disorders in cerebral function could be identified by psychological testing which were the "result" of epilepsy and which "predisposed" to the development of psychosis has been mentioned above. The features of the psychosis are consistent with the previous descriptions reviewed above, emphasizing the lack of

affective flattening, the extreme religiosity and the organic symptoms and signs in thought processes. He also noted that:

> Many of the patients were concerned with the clarity of their thinking and made significant efforts to control, restrict and contain emotions and actions in order to become clear, accurate and realistic.

Glaser also points out that if the psychosis is related to epilepsy, it may be altered by successful treatment of epilepsy, which is indeed observed in some but not all cases.(34)

Flor-Henry also studied only patients with temporal lobe epilepsy, but divided them into 50 with psychosis and 50 without psychosis. It might be mentioned that his material was drawn from the records of the same hospital as that of Slater and Beard, over a 15 year period that overlapped ten of the twelve years of the previous study. Comparing psychotics to controls, it became apparent that psychotics had significantly fewer seizures, particularly with reference to psychomotor and minor temporal seizures (by which are meant what I have called temporal lobe seizures). Stating that the laterality of EEG foci was about the same for the whole group, he noted that there was a significant excess of dominant-hemisphere (left-sided) EEG foci in the psychotic group, which became even more significant when the bilateral foci group was added, suggesting that the involvement of the dominant hemisphere was associated with the psychosis.

The form of the psychosis was schizophrenic in 21, schizoaffective in 11, manic-depressive in 9 and of a confusional nature in 9. The numbers were too small to allow statistical interpretation, but it was seen that manic-depressive and schizoaffective patients were different from schizophrenic and confusional patients, in that the affective patients tended to have better psychosocial adjustment, greater intelligence, and involvement of the non-dominant hemisphere with focal epileptic activity. There was a trend in which manic-depressives had fewer temporal lobe and more major (grand mal) seizures than the schizoaffective cases, and similarly through the confusional cases to the schizophrenics, who had the most temporal lobe seizures and the most total seizures. In a footnote, he briefly mentions six centrencephalic epileptics with psychosis, who (compared to the temporal lobe epileptics) had brief, confusional psychoses, and tended to have lower intelligence and a more disturbed environment. This is similar to the findings of Dongier (28). Finally, he concludes that the psychoses of temporal lobe epilepsy are not so much "organic" in the general, non-specific sense implied by Slater and Beard, as they are "epileptic" psychoses fundamentally related to seizures more than to brain damage, and seems to favor the idea stated by Symonds above (68) of an "epileptogenic disturbance of function" causing epilepsy and

psychosis (36).

It must be pointed out that the only statistically significant results of Flor-Henry's study are that the psychotics have fewer seizures, particularly temporal lobe seizures, and have more involvement of the dominant hemisphere with an EEG focus, than do controls with temporal lobe epilepsy and no psychosis. Other findings are open to question, and the question of "epileptic" psychosis can only be called a speculative issue at present.

It is pertinent to note here an issue which Flor-Henry feels to be critical, namely the relationship between psychosis and seizure frequency or psychosis and EEG activity. Landolt first noted this phenomenon of improvement in EEG sometimes to normality during a psychotic episode, which he called "forced normalization." In Dongier's study, focal or synchronous discharge disappeared during the psychotic episode in 24.4%, and there was improvement in the EEG in another 5%. There was no significant correlation to type of epilepsy or type of psychosis (28). Slater and Beard found normalization of the EEG in only 3 of their 69 cases (66), and Glaser found in his 37 cases, two with normal and four with an improved EEG (34). The claimed inverse relation between seizures and psychosis was noted by Gibbs (37) and by Pond (10), who qualified the idea with the statement that it only held for certain patients in certain stages of their psychosis. Flor-Henry

demonstrated this relationship for his cases (36), but Slater and Beard (66) and Glaser (34) do not support this idea. A recent comparison of spiking in sphenoidal electrodes in psychotic as opposed to non-psychotic temporal lobe epileptics found spiking to be more common among the psychotic group, but the relation of this to scalp EEG recordings in the past, and to observed seizures, is still unclear (69).

Patients with psychosis who undergo temporal lobectomy were initially claimed to be improved mentally if the seizures were controlled (70,71). Later studies showed that the best results were in patients with acute confusional psychoses, intermediate results in those with affective or schizoaffective psychoses, and the worst in those with schizophrenia-like psychoses (72,73), suggesting that the relation to epileptic activity is greatest for those in whom the post-operative result is best (see also Chapter Five).

F. "Are Psychomotor Epileptics Different?"

The title for this section comes from an article by Small et al. (19) from the laboratory of Stevens, who has published other articles with a similar point of view (20,50). The essence of her argument is as follows:

> In addition to the poorly understood relationship between psychiatric disorders and epilepsy, there is a second and equally interesting question, i.e., whether temporal lobe-psychomotor epilepsies are associated with a special predilection for psychopathology compared to "non-temporal" epilepsies. If, as indicated by Gastaut, Mignone et al., as well as our own studies, between 55% and 65% of individuals with a seizure disorder over the age of 30 have psychomotor or temporal lobe epilepsy, the incidence of psychomotor epilepsy inevitably exceeds that of other epilepsies among any group of adults with seizures and psychosis. (20)

She notes that sampling bias is particularly bothersome in assessing prevalence of temporal lobe epilepsy, and that one source of such bias is a public facility to which those who are poor and in inadequate seizure control will tend to go.

In the social studies reported above, it was noted that those in the lowest social class among epileptics were those with the most frequent seizures. Temporal lobe epilepsy is notorious for being difficult to control, as Stevens and many others have written. Thus public facilities such as university hospital clinics will be particularly susceptible to bias in having an excess of temporal lobe epileptics among their

population of epileptics served.

Of the studies cited by Stevens above, hers is drawn from such a university hospital clinic population. That of Gastaut does not give a description of the source of the patients, but places the prevalence of temporal lobe epilepsy among all epileptics at 30% of in-patients and 80% of out-patients (74). Other studies by Gastaut, cited by Dongier (28), place the prevalence at 62.7%, but again no information is given as to the nature of the population. I suspect it is drawn at least partly from the university hospital clinic with which Professor Gastaut has association, in Marseilles. The third study cited by Stevens is that of Mignone et al., who gathered patients from the psychiatric and neurosurgical services of the National Institute of Neurological Diseases and Stroke. 65% of their cases had temporal lobe epilepsy (55), but again the nature of the source makes the general validity of this as a prevalence figure highly questionable. Psychiatrists and neurosurgeons are particularly likely to see temporal lobe epileptics, and those they see will be particularly impaired.

Two other studies are cited by Stevens and her group as supporting a 55-65% prevalence figure, in a different article (50). Both the studies of Guerrant et al. (51) and that of Vislie and Henrikson (52) were unavailable to me for review,

but other literature that describes them points out the university hospital clinic origin of their study population.

Jasper et al. state that for the general epileptic population, the prevalence of temporal lobe epilepsy is about one in five or 20% (75). This is the same figure given by Lennox for his private patients (76), although it is greater with older patients (34% in those over 40 years). Finally, the survey of general practices in Britain by Pond and Bidwell found 245 epileptics of whom 39 were temporal lobe epileptics, or 16% (8). Although the figure of Lennox is skewed by his position as a consultant neurologist, and that of Pond and Bidwell may be skewed in that temporal lobe epileptics might gravitate to special centers or institutions rather than general practices, it seems reasonable to conclude that the prevalence of temporal lobe epilepsy in all epilepsy is somewhere between one-fourth and one third of cases.

This figure is very important, as I have noted above, in assessing the relationship between temporal lobe epilepsy and psychopathology. Many studies have found a figure greater than one-third for the prevalence of temporal lobe epilepsy among epileptics with psychopathology (11,28,29,65,66), including those of Stevens and her group (19,20,50) and those of the studies she cites in support of her idea (51,52,55). Although clearly not all of these studies are equally valid

as a source of prevalence, certainly the aggregate strongly supports the idea of an excess of temporal lobe epilepsy among epileptics with psychopathology. The figures on prevalence of psychosis in epileptics and in temporal lobe epilepsy, stated above under "Psychosis," also support this idea.

The second important question that must be answered before the question that is the title of this section can be answered, is: "What differences are there between the psychopathology of temporal lobe epilepsy and that of other forms of epilepsy?" I have attempted to answer this in the sections "Causes of Psychopathology" and "Personality Studies" above. It will be recalled that, due in large measure to the carefully controlled studies of Stevens and her group (19,20) and those of others (esp. 55), no difference can be found consistently between the psychopathology of these two groups. The suggestion of Glaser that certain cerebral deficits exist and predispose to psychopathology probably implies that these deficits occur in other epileptics, inasmuch as the observed result (psychopathology) cannot be differentiated. Possible reasons for this are mentioned in "Personality Studies."

Thus I conclude that temporal lobe epileptics are indeed different in that they have more of the same psychopathology

which is shown by all epileptics. In this I am supported by the literature. I suggest further that a reason contributing to this is the involvement of the temporal lobe and limbic structures in abnormal excitation and the resultant disturbance of perceptions, an involvement probably shared by the generalized epilepsies. I suspect a Gallup Poll of neurologists across the country would support me in this latter suggestion.

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## MATERIALS AND METHODS

### A. Patients

Fifty-nine patients with temporal lobe seizures as described in the review above were seen in the Seizure Clinic or the Clinical Research Unit (Hunter 5) of the Yale-New Haven Hospital Medical Center. Patients on the Clinical Research Unit were admitted electively for study and do not necessarily represent patients with more severe difficulties. An additional four patients were seen in the Seizure Clinic of the West Haven Veteran's Administration Hospital.

Sufficient for entry into the study were the presence of the following:

1. Presence of temporal lobe seizures;
2. Anticipated ability to convey required information, either verbally (to me) or in written form;
3. Existence of adequate records to document medical and neurological history.

All patients were questioned personally as to the nature of their seizures, and the impression obtained compared with that of neurologists responsible for their care. The conversation was also directed to ascertain the patient's willingness to complete the study and his ability to do so. No coercion was ever applied.

All patients arriving for routine visit to the Seizure Clinic at Yale-New Haven Hospital between April and

October of 1972, except for brief intervals when the examiner was occupied elsewhere, were considered as possible candidates for the study. All those fulfilling the three criteria outlined above were requested to join the study. There were several individuals who declined, but most were willing to cooperate.

The patient's role in the study consisted of three parts:

1. Providing requested social, psychological and neurological data to the examiner, which was then checked against the written medical record and the opinion of the physicians responsible for his care;
2. Cooperating in the administration of a mental status examination which was coded into a form (KDS-5) to be described below;
3. Completion of three self-scored questionnaires (KDS-1, KDS-2, KDS-3A), described below, which were then returned to the examiner.

The time required for each patient to perform these three tasks varied from approximately one-half to three hours.

B. Social, Psychological and Neurological Information

Questions were put to the patient and the responses recorded according to the following scheme.

1. Social--The patient's present age and sex were noted. His racial background (Caucasian or Negro) was noted. His educational level was determined by investigating whether he had completed high school, and if he had, whether he had continued his education in college or training school. Occupational status was determined as either full-time, part-time or no employment. Socio-economic status was estimated from knowledge of income, educational level and occupational status, and classed as either upper, middle or lower class. The disturbance of the patient's social functioning was determined according to the following criteria:

No disturbance

Mild disturbance---noted only by the patient

Moderate disturbance---noted by both the patient and other observers

Incapaciting disturbance---patient is unable to function in the way he wants and is accustomed to functioning

2. Psychological--The patient was asked whether or not he had ever been in psychotherapy or was now in psychotherapy, omitting attendance at a medication clinic without other therapy. He was also asked whether he had ever been hospitalized in a psychiatric hospital or for psychiatric

reasons

reasons, and if so, how many times he had been so hospitalized.

3. Neurological--The following information was obtained either from the patient, his physician or the record.

1. Electroencephalogram (recent, within the past year): classed by technique as either routine scalp EEG while sleeping; or routine scalp EEG while waking; classed by findings as normal, borderline, diffusely abnormal, left temporal focus, right temporal lobe focus, bilateral temporal lobe foci, any other focus; classed not available if none was available.
2. Type of Onset: classed as infectious, traumatic, surgical, due to alcohol, or unknown.
3. Age at onset was noted and duration of epilepsy determined from this and the present age.
4. The presence or absence of a family history of seizures was noted, and if present, classed as either immediate or remote family.
5. The presence or absence or neurological signs found by a neurologist was noted, and if present, classed as motor only or motor plus mental.
6. The history of seizure control was noted, and classed as either stable for years, intermittently good and bad, or stormy and never satisfactory.

7. The history of seizure types was noted, and classed as either temporal lobe only, temporal lobe plus grand mal, or temporal lobe plus other types.
8. The seizure frequency was recorded for temporal lobe seizures in the past two or three months, and the result classed by the following:

   More than one a day

   One a day

   Ten to twenty-nine a month

   One to nine a month

   One to eleven a year

   Less than one a year
9. The drugs and the dosages which the patient was taklng were noted and recorded. All drugs, including anticonvulsants, psychotropics and medical drugs were noted.

It was determined to classify seizures according to whether they were grand mal, temporal lobe or other, rather than use the International Classification proposed by Gastaut (see above), because the manifestations of each patient's seizure often included several of the entities in that classification, and in all cases direct observation of each patient's seizure would have been necessary to classify him accurately.

C. Questionnaires

The questionnaires completed by the patient included KDS-1, KDS-2, and KDS-3A, which are appended as Appendix 1. KDS-1 is a 41-item form requesting yes or no responses to questions concerning the patient's present feelings and attitudes. KDS-2 is a 64-item form requesting yes or no responses to questions concerning the patient's present symptoms. KDS-3A is a 45-item form requesting yes or no responses to questions concerning how the patient generally reacts or feels in a "chronic" sense, and is thus an index of personality.

The questionnaire completed by the examiner was KDS-5, which is appended with the other questionnaires as Appendix 1. This is an extensive survey of the patient's presenting features as observed by the examiner, as well as a standardized mental status examination encompassing most of the usual features of orientation, general knowledge, understanding, digit retention, mental calculation, serial sevens and presence or absence of perseveration.

The use of the questionnaires has been reported in several previous publications (1-5). The findings from their use in psychiatric patients, as well as those from their use in normal people, will be presented by comparison with the patients of this study in the Results section. The data on which this comparison will be based, in preparation for publication (6), have been collected in two groups of people:

1. Fifty normal people who had never sought psychiatric help as inpatients or outpatients. None suffered from any medical illness or was taking any medications. The mean age and the social class were highly similar to the group studied here.
2. One hundred and thirty three patients with primary affective disease as the presenting complaint, corrently in remission, were tested as outpatients attending a psychiatric clinic or medication clinic. All were on drugs, including lithium (86) and tricyclics or monoamine oxidase inhibitors (47). The mean age and the social class were roughly similar to the group studied here.

Computer processing of the data in the questionnaires was done by the Yale Computer Center. Each patient was scored with regard to how many points he achieved on each of seventeen scales, which are appended as Appendix 2. For each response on KDS-1 and KDS-2, weight was given as noted in the scales, and a score recorded for the symptom clusters named: Anxiety (A), Depression (D), Psychosis (P), Organicity (O), Mania (M), Neurosis (N), Impulsivity (I), Paranoia (PA) and Suicidal Ideation (SU). The remaining symptom clusters were similarly derived from KDS-3A: Chronic Anxiety (A3), Chronic Depression (D3), Chronic Impulsivity (I3), Chronic Neuroticism (N3), Chronic Paranoia (PA3), Personality Disorder (PER), Chronic Psychopathy (PSY) and Chronic Suicidal Ideation (SUI).

The scales for the symptom clusters quantify symptoms commonly associated with the entity whose score they determine, and the higher the score for each entity, the more severe the disorder. Inasmuch as the scales are arbitrarily determined to reflect clinical experience, the scores on these items should only be interpreted in terms of the questions which comprise the scale and not in terms of the name which is used to refer to it. Whether or not there is a direct relationship between the questions in each scale and the name affixed to the scale is not susceptible to proof at this time.

D. Analysis

Correlations between social, psychological and neurological data and the scores on the symptom clusters were performed with the assistance of the Yale Computer Center and the Research Unit of the Connecticut Mental Health Center. Statistical tests of significance used included the correlation coefficient, the t test, and the chi-square test.

It is apparent from perusal of the scales for each symptom cluster that the scales share some items in common, i.e., a "Yes" response will raise the score for each scale using that item. I have calculated the number of points each scale shares with every other scale in the determination of scores for symptom clusters, presented as Table M-1. The percentage of the total that this represents is the extent to which the scores for the two scales will be raised together, if there is no actual association in the patient between the two symptom clusters. Abbreviations used are as described in the previous section.

Table M-1

Points Shared in Common Between Symptom Clusters

| Cluster | A | D | P | O | N | M | I | PA | SU |
|---|---|---|---|---|---|---|---|---|---|
| A | -- | 3 | 0 | 6 | 2 | 2 | 2 | 0 | 9 |
| D | 3 | -- | 0 | 18 | 1 | 3 | 0 | 0 | 20 |
| P | 0 | 0 | -- | 1 | 1 | 0 | 0 | 13 | 1 |
| O | 6 | 18 | 1 | -- | 0 | 0 | 2 | 0 | 9 |
| N | 2 | 1 | 1 | 0 | -- | 2 | 0 | 1 | 0 |
| M | 2 | 3 | 0 | 0 | 2 | -- | 6 | 1 | 4 |
| I | 2 | 0 | 0 | 2 | 0 | 6 | -- | 1 | 2 |
| PA | 0 | 0 | 13 | 0 | 1 | 1 | 1 | -- | 0 |
| SU | 9 | 20 | 1 | 9 | 0 | 4 | 2 | 0 | -- |
| Maximum Score | 48 | 55 | 29 | 45 | 13 | 26 | 14 | 16 | 32 |

| Cluster | A3 | D3 | I3 | N3 | PA3 | PER | PSY | SUI |
|---|---|---|---|---|---|---|---|---|
| A3 | -- | 1 | 0 | 0 | 0 | 0 | 0 | 6 |
| D3 | 1 | -- | 0 | 0 | 1 | 1 | 0 | 2 |
| I3 | 0 | 0 | -- | 0 | 0 | 0 | 3 | 7 |
| N3 | 0 | 0 | 0 | -- | 0 | 3 | 0 | 2 |
| PA3 | 0 | 1 | 0 | 0 | -- | 2 | 0 | 1 |
| PER | 0 | 1 | 0 | 3 | 2 | -- | 0 | 5 |
| PSY | 0 | 0 | 3 | 0 | 0 | 0 | -- | 3 |
| SUI | 6 | 2 | 7 | 2 | 1 | 5 | 3 | -- |
| Maximum Score | 13 | 6 | 15 | 15 | 7 | 23 | 9 | 23 |

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# RESULTS

## A. Distributions of Measured Parameters

The results of the social, psychological and neurological parameters are presented in Table R-1. This represents the distribution of these parameters in the entire group of 63 temporal lobe epileptics. The results of the examiner's appraisal and mental status examination are presented in Table R-2. This is the distribution of these items for all patients tested; as five were not personally examined, the total for Table R-2 is 58. The small numbers involved in Part A of Table R-2 were not further analyzed, as the groups are too small to be significant.

The mean scores for all symptom clusters are given in Table R-3, together with the medians, standard deviations and ranges. The actual distribution of the scores is appended as Appendix 3. The significance and correlations of these data will be discussed below.

Table R-1

A. Social Parameters

Sex
- Male----------25
- Female----------38

Work
- None----------41
- Part-time----------3
- Full-time----------13

Race
- Caucasian----------59
- Negro----------4

Education
- Did not finish high school--------33
- Presently high school student------3
- High school graduate----------21
- College or training school---------6

Socioeconomic Class
- Lower----------17
- Middle----------44
- Upper----------2

Social Functioning
- No disturbance----------3
- Mild (noticed only by patient)----27
- Moderate(also noticed by others)--26
- Incapacitating----------7

B. Psychological Parameters

History of Psychotherapy
- Never in therapy----------32
- Recently in therapy----------14
- Currently in therapy----------17

History of Psychiatric Hospitalization
- Never hospitalized----------46
- Hospitalized once only----------4
- Hospitalized more than once-------13

C. Neurological Parameters

Type of Onset
- Infection----------6
- Trauma----------14
- Surgery----------1
- Alcohol----------1
- Unknown----------41

Table R-1 (Cont.)

Age at Onset
- Mean---15.238
- Median---14.000
- S.D.---10.560
- Range---1 to 47

Present Age
- Mean---38.379
- Median---40.000
- S.D.---21.073
- Range---17 to 71

Duration of Epilepsy
- Mean---21.444
- Median---20.000
- S.D.---15.340
- Range---2 to 67

Electroencephalogram
- Routine waking---56
- Routine sleeping---3
- Not available---4

- Normal---5
- Borderline---5
- Diffusely abnormal---20
- Left temporal focus---11
- Right temporal focus---6
- Bilateral temporal foci---10
- Other foci---2

Seizure Frequency
- More than one a day---4
- One a day---4
- Ten to twenty-nine a month---7
- One to nine a month---31
- One to eleven a year---12
- Less than one a year---5

History of Seizure Control
- Stable for years---15
- Intermittent good and bad---29
- Stormy, never good---19

Types of Seizures
- Temporal lobe only---21
- Temporal lobe plus grand mal---37
- Temporal lobe plus other type---5

Family History of Seizures
- None---54
- Immediate family---8
- Remote family---1

Table R-1 (Cont.)

Neurological Signs
- None----------49
- Motor only----------9
- Motor plus mental----------5

Table R-2

A. Presentation

Appearance
- Normal----------54
- Unusually unattractive----------1
- Visible physical deformity----------3

Dress
- Acceptable----------54
- Meticulous----------1
- Careless----------1
- Bedridden----------2

Weight
- About right----------48
- Overweight----------10

Motor Activity
- Bedridden----------2
- Graceful----------46
- Clumsy----------6
- Unusual gait----------3
- Clumsy and unusual gait----------1
- Choreiform movements----------1
- Tics----------1
- Keeps still most of the time----------1

Speech
- Easily understandable----------51
- Word-finding difficulty----------2
- Monotonous----------1
- Slurred----------4

Sensory Systems
- Normal----------56
- Markedly impaired vision----------1
- Deaf----------1

Table R-2 (Cont.)

Attitude
- Friendly----55
- Fearful----1
- Sarcastic----2

Personal Observations
- Lack of spontaneous non-verbal expression----3
- Lack of emotional expression or response----2
- Feelings of sadness, worthlessness, etc.----4
- Observable manifestations of anxiety/panic----1
- Verbal expression of overt hostility----1
- Decreased motor activity----5
- Increased motor activity----1
- Disorganized speech----1
- Suspicious and distrustful----1
- Concerned with suicide----2
- Drowsy, inattentive----1
- Intermittently alert and drowsy----1
- Distinct memory impairment----3
- Little or no spontaneous speech----1
- Preoccupied with physical health----4
- Afraid of specific objects or situations----2
- Repeats certain acts or thoughts----2
- Skips from one subject to another----1
- Avoids eye contact----3

B. Mental Status Examination

| Question | Correct | Incorrect |
|---|---|---|
| What is the name of this place? | 58 | 0 |
| What is today's date? | 51 | 6 |
| What are the names of the past three presidents of the U.S.A.? | 46 | 12 |
| **Now:** count from 1 to 23. | 58 | 0 |
| What are the colors in the flag? | 57 | 1 |
| What is a thermometer? | 50 | 8 |
| How far is it from L.A. to New York? | 35 | 23 |
| Name three countries in the Middle East. | 16 | 42 |
| Contilnu counting from where you left off. | 53 | 5 |

Serial Sevens
- Perfect----14
- One error----17
- Two errors----8
- Three errors----2
- Four errors----0
- Five errors----2
- Cannot start it----15

Perseveration
- Present----12
- Absent----46

Table R-3

Scores on Symptom Clusters

| Scale | Mean | Median | S.D. | Range |
|---|---|---|---|---|
| Anxiety | 13.578 | 14.000 | 8.511 | 0-33 |
| Depression | 20.250 | 19.000 | 10.279 | 0-43 |
| Psychosis | 6.344 | 4.000 | 6.883 | 0-29 |
| Organicity | 16.500 | 17.000 | 9.069 | 0-36 |
| Neurosis | 4.250 | 3.000 | 3.504 | 0-12 |
| Mania | 9.672 | 10.000 | 4.334 | 1-20 |
| Impulsivity | 5.484 | 5.000 | 3.444 | 0-14 |
| Paranoia | 4.063 | 3.000 | 4.293 | 0-16 |
| Suicidal Ideation | 10.813 | 11.000 | 6.656 | 0-27 |
| Chronic Anxiety | 6.406 | 6.500 | 3.512 | 0-13 |
| Chronic Depression | 3.438 | 3.500 | 2.007 | 0-6 |
| Chronic Impulsivity | 5.016 | 5.000 | 3.393 | 0-15 |
| Chronic Neuroticism | 4.797 | 5.000 | 3.645 | 0-15 |
| Chronic Paranoia | 2.828 | 2.500 | 1.728 | 0-7 |
| Personality Disorder | 10.250 | 10.000 | 3.929 | 0-18 |
| Chronic Psychopathy | 2.313 | 1.500 | 2.135 | 0-7 |
| Chronic Suicidal Ideation | 8.641 | 9.000 | 4.438 | 0-21 |

B. Correlations Between Measured Parameters

Correlation coefficients between data of linear variation was obtained by construction of a correlation matrix, the results of which are given as Appendix 4. Results of this analysis that proved to be statistically significant or interesting were scrutinized and are discussed below.

Table R-1, Part A, reveals that a substantial portion of the group studied is in the lower social class, out of work and did not finish high school. An attempt to understand the interrelationships of these items was made by investigating the correlations between several of the parameters. The results of this investigation are presented in Table R-4, with the test of significance and the result. Except for a preponderance of people working full-time in the group of middle social class, no relationship emerges from this analysis that could not have arisen by chance alone.

1. Internally Consistent Correlations--Age of onset and duration of seizures measure the same entity, and one is derived from the other, so their correlation is very highly significant ($p < .001$). History of seizure control and present seizure frequency also measure a similar entity, the number of seizures suffered over time, and this correlation is also very highly significant ($p < .001$). The meaning of this statement is that for the group studied, the present seizure frequency is a reliable predictor of the degree of seizure control achieved over the duration of epilepsy. The

Table R-4

Relations of Social Parameters to Other Parameters

| Relationship sought | Chi-square | Significance |
|---|---|---|
| Work versus sex | 1.00 | p=0.30(NS)* |
| Not working versus social class | 2.10 | p=0.15(NS) |
| Fulltime work versus middle class | 4.87 | p<0.05(Sig.)** |
| Work versus education | 0.40 | p=0.50(NS) |
| Work versus social functioning | 2.00 | p=0.60(NS) |
| Work versus history of control | 0.60 | p=0.45(NS) |
| Education versus social class | 0.70 | p=0.40(NS) |
| Education versus age of onset | 1.50 | p=0.20(NS) |
| Education versus duration | 2.60 | p=0.10(NS) |
| Education versus mental status examination (all items) | (No significant correlation) | |

*NS=Not Significant

**Sig.=Significant

relation of a history of psychotherapy and a history of psychiatric hospitalization ($p<.001$) is also measuring a related entity, the severity of psychopathology, and suggests as a very highly significant correlation that one cannot distinguish between patients with varying degrees of psychopathology in this group on the basis of the psychiatric history, as those with a history of one mode are most likely to give a history of the other mode. Finally, the very highly significant negative correlation between phenobarbital dosage and primidone dosage ($p<.001$) reflects the guidelines followed in the Seizure Clinic, that whenever possible patients are placed on one or the other, but not both, of these drugs.

2. Social Class and Social Functioning--The correlations involving social class are interpreted as, the higher the cocioeconomic status, the lower the score correlated to it. Symptom cluster scores associated in this way include those named Suicidal Ideation, which is highly significant ($p<.01$) and which is an association well known to those who work with suicidal patients. It is thus a confirmation of a known relationship. The other symptom cluster scores which are significantly correlated ($p<.05$) with socioeconomic status are those named Anxiety, Psychosis, Neurosis and Chronic Paranoia, all of which are lower the higher the social class. It must be recalled that the determination of socioeconomic status and social class was not more precise than the assignment by those responsible for the patient's care of this parameter, based on education, income and occupation.

Thus the assignment must be considered subjective and subject to observer bias.

Two correlations emerge as determinants of the degree of disturbance of social functioning. A history of psychiatric hospitalization is significant ($p<.05$) in the worsening of social functioning as measured, and a greater frequency of seizures is highly significant ($p<.01$) in the worsening of social functioning. Both of these relationships reflect the difficulties of living in society with the problems of epilepsy and significant psychiatric disturbance as independent parameters. It will be noted that no correlation emerges between seizure frequency and a history of psychiatric hospitalization.

3. Seizure Frequency--The frequency of seizures was correlated with the history of seizure control and the degree of disturbance of social functioning, as noted above. On the mental status examination, seizure frequency was significantly correlated with the presence of perseveration as observed by the examiner. The direction of the relationship was such that those with less than one seizure a month were significantly more likely ($p<.05$) to demonstrate perseveration than those with more than one seizure a month. The reason for this is obscure, but may reflect the chronicity of epilepsy. No correlation emerged between seizure frequency and a history of traumatic onset to suggest that those more likely to perseverate were those with a greater likelihood of significant brain damage. However, there was a suggestion,

not statistically significant, of a relationship between the presence of perseveration and a history of traumatic onset ($p=.09$).

The relationship between seizure frequency and the scores on the symptom cluster scales is of great importance and will be discussed further in the Discussion. The values for this correlation are given in Table R-5, in which the mean scores for each subgroup of the seizure frequency are given for each symptom cluster scale. It is apparent that certain scores, those for Organicity, Suicidal Ideation and Chronic Suicidal Ideation, are highly significantly related to seizure frequency; those for Anxiety, Chronic Anxiety, Depression, Mania and Impulsivity are significantly related ($p<.05$) to seizure frequency; and those for all other symptom clusters, including Psychosis and six of the eight scales attempting to reflect "chronic" difficulties of a psychiatric nature, are unrelated to seizure frequency. The meaning of this finding will be considered below.

4. Type of Onset--The etiology of epilepsy was seen to be significantly related to the age of onset ($p<.05$), as is evident from Table R-6.

Table R-6

| Age of Onset | Infection | Trauma | Surgery | Alcohol | Unknown | Total |
|---|---|---|---|---|---|---|
| 1-10 | 2 | 3 | 0 | 0 | 14 | 19 |
| 11-15 | 1 | 1 | 0 | 0 | 17 | 19 |
| 16-25 | 2 | 6 | 0 | 1 | 7 | 16 |
| 26-35 | 1 | 3 | 0 | 0 | 1 | 5 |
| 36-50 | 0 | 1 | 1 | 0 | 2 | 4 |
| Total | 6 | 14 | 1 | 1 | 41 | 63 |

Table R-5

Seizure Freq. vs. Mean Scores (Sig.)

| Seizure Freq. | O | SU | SUI | A | A3 | D | M | I |
|---|---|---|---|---|---|---|---|---|
| 1/yr. | 6.8 | 2.6 | 5.4 | 7.6 | 4.2 | 9.0 | 7.0 | 3.0 |
| 1-11/yr. | 14.0 | 8.8 | 7.4 | 11.8 | 4.7 | 16.5 | 9.8 | 4.7 |
| 1-9/mo. | 17.7 | 11.9 | 9.5 | 14.3 | 7.6 | 22.2 | 8.9 | 5.7 |
| 11-29/mo. | 15.2 | 9.9 | 6.6 | 10.7 | 5.4 | 19.4 | 10.4 | 5.1 |
| 1/day | 24.3 | 16.8 | 13.0 | 19.8 | 9.8 | 27.0 | 14.8 | 8.5 |
| 1/day | 20.0 | 7.0 | 13.5 | 17.7 | 7.0 | 23.7 | 11.5 | 6.5 |
| r | .329 | .341 | .385 | .246 | .263 | .316 | .284 | .268 |
| p | .01 | .01 | .01 | .05 | .05 | .05 | .05 | .05 |

| Seizure Freq. | PA3 | I3 | N3 | PSY | D3 | N | PER | P | PA |
|---|---|---|---|---|---|---|---|---|---|
| 1/yr. | 2.4 | 3.8 | 2.4 | 1.8 | 3.0 | 1.2 | 8.2 | 1.0 | 0.2 |
| 1-11/yr. | 2.3 | 5.5 | 4.6 | 1.5 | 2.7 | 4.9 | 8.3 | 7.3 | 5.0 |
| 1-9/mo. | 3.1 | 4.7 | 5.4 | 2.8 | 3.9 | 4.1 | 11.8 | 6.9 | 5.0 |
| 11-29/mo. | 2.1 | 3.7 | 2.6 | 7.1 | 2.4 | 3.4 | 9.0 | 2.3 | 2.4 |
| 1/day | 3.0 | 7.8 | 7.0 | 3.8 | 4.3 | 6.5 | 9.8 | 7.8 | 3.5 |
| 1/day | 4.8 | 7.5 | 6.8 | 3.8 | 4.8 | 5.0 | 11.8 | 10.0 | 5.5 |
| r | .212 | .196 | .194 | .193 | .171 | .167 | .158 | .126 | .078 |
| p | NS | NS | NS | NS | NS | NS | NS | NS | NS |

When the mean age of onset for those with traumatic etiology was compared with the mean age of onset of those with unknown etiology, using the t test, a highly significant ($p<.01$) difference emerged, with the unknown group having a younger age of onset. No significant difference emerged from a similar conparison of mean age of onset of those with infectious and unknown etiology, probably due to the small size of the group with an infectious etiology (N=6).

Examination of the relationships between type of onset and scores on symptom cluster scales revealed a general trend for those with traumatic onset to have higher scores than those with unknown onset. This was only significant ($p<.05$), using the t test, for the scales named Depression, Psychosis, and Chronic Neuroticism, and reflects a common awareness mentioned in the review above that greater intellectual impairment and more severe psychopathology are seen in patients with a history of trauma and brain damage.

It is interesting that examination of the relationship between a history of traumatic onset and presence of neurological signs did not reveal a statistically significant correlation, nor did examination of the relationship between a history of unknown onset and a family history of seizures. Thus these determinants could not be confirmed in this study.

5. Electroencephalogram--As mentioned in the review above, not all temporal lobe epileptics will demonstrate temporal lobe foci on routine scalp EEG. With this technique, five patients

had normal tracings and five borderline tracings; the rest were abnormal, and in 27 cases demonstrated temporal lobe foci. Examination of the relationship between the laterality of the EEG focus and the scores on symptom cluster scales, using the t test, revealed correlations that were not always consistent. Depression scores were significantly higher in those with right temporal EEG foci than those with bilateral temporal EEG foci ($p<.05$), but not significantly different from those with left temporal EEG foci. Psychosis scores were significantly higher in those with left temporal EEG foci than those with bilateral EEG foci ($p<.05$), but not significantly different from those with right temporal EEG foci. Patients with a right temporal EEG focus had significantly higher scores for Organicity than those with bilateral EEG foci ($p<.05$), for Impulsivity than those with bilateral EEG foci ($p<.001$), and for Chronic Neuroticism than those with left temporal foci ($p<.05$). The low level of significance, the relatively small sizes of the groups, and the patchy distribution of correlations in ways that do not always correspond to inferences about cerebral dominance, lead me to impute little meaning to these findings. However, those for the Depression and Psychosis scores tend to support others' contentions and will be discussed further.

6. Therapy--Patients with a history of psychotherapy were significantly likely to have a higher score for the Neurosis scale ($p<.05$); the psychiatric history was not otherwise correlated to the scores on the symptom cluster scales.

Patients on a higher dosage of primidone were unlikely to be on a high dose of phenobarbital, as mentioned. These patients also had significantly fewer seizures ($p<.05$), a significantly better history of seizure control ($p<.05$), and were more likely to have had an early onset of seizures ($p<.05$). The relationship between seizure frequency and history of seizure control has been mentioned above. The other relationships appear to reflect either the usage of primidone in the clinic or aspects of the drug itself in the therapy of epilepsy.

7. Duration--It was noted above that duration was related to age of onset, as would be expected. There was no correlation between the score on the Psychosis score and duration of epilepsy, as has been claimed by others and will be discussed further. Patients with a longer duration of epilepsy tended to have significantly lower scores on the scales for Chronic Impulsivity and Chronic Suicidal Ideation, ($p<.05$), a relationship which undoubtedly reflects the adaptations to chronic disease present for many years.

8. Correlations Sought and Not Found--In addition to those correlations attempted and described above, several others were felt to be of interest and examined. There was not an excess of patients with a greater number of seizures in lower social class. Although disturbance of social functioning was seen to correlate with a history of psychiatric hospitalization, no significant relationship was found between social functioning disturbance and a history of psychotherapy. The electroencephalogram might have been

expected to differ with respect to types of seizures or the presence of neurological signs. There was a trend for patients with diffusely abnormal EEG tracings to have both temporal lobe and grand mal seizures, but it was not statistically significant ($p=.10$). There was also a trend for patients with neurological signs to have demonstrable temporal lobe EEG foci on routine scalp tracings, but again this was not statistically significant ($p=.06$).

C. Comparisons with Control Groups

The mean scores for both groups described in the Materials and Methods section were compared with the mean scores for the patients with temporal lobe epilepsy for all the symptom cluster scales as defined above. The results of this comparison are given in Table R-7; using the t test, the value for t and the probability of the difference arising by chance (p) were determined and are shown. In this table, T.L.E. refers to patients with temporal lobe epilepsy and "affectives" ("aff.") are those patients with primary affective illness in remission under treatment as described above. "Norm." refers to normal people.

It is readily apparent that on virtually every scale measured, the temporal lobe epileptics scored higher than both the normal population and the patients with primary affective illness in remission. This difference was highly significant. It must be recalled that only a small number of the temporal lobe epileptics were on psychotropic or antidepressant medication: two on Elavil, three on Valium, three on Librium, one on lithium, and seven on a variety of phenothiazines including Stelazine (2), Trilafon (3), Prolixin (1) and Mellaril (1). In most cases the dosage was relatively low and in many was on a "take only when needed" basis. Inspection of the scores of these patients reveals that, with one exception, they are as high or higher than the mean scores for the group. Many of these patients have a psychiatric history. One might conclude that patients with more severe disturbance were not controlled by their psychotropic drugs.

Table R-7

Comparison of T.L.E. with Affectives and Normals

| Cluster | t(p) Aff. vs. Norm. | t(p) T.L.E.vs. Norm. | t(p) T.L.E.vs Aff. |
|---|---|---|---|
| A | 3.643 (<.001) | 8.845 (<.001) | 5.930 (<.001) |
| D | 0.664 (NS) | 5.873 (<.001) | 5.150 (<.001) |
| P | 1.192 (NS) | 4.455 (<.001) | 3.987 (<.001) |
| O | 1.786 (<.05) | 7.048 (<.001) | 5.113 (<.001) |
| N | 1.112 (NS) | 4.934 (<.001) | 4.893 (<.001) |
| M | -6.395 (<.001) | -1.149 (NS) | 5.645 (<.001) |
| I | -1.481 (NS) | 3.993 (<.001) | 6.456 (<.001) |
| PA | -0.802 (NS) | 3.218 (<.001) | 4.601 (<.001) |
| SU | 3.132 (<.001) | 7.538 (<.001) | 4.801 (<.001) |
| A3 | 0.427 (NS) | 2.483 (<.01) | 2.624 (<.01) |
| D3 | -0.158 (NS) | 3.652 (<.001) | 3.910 (<.001) |
| I3 | 0.439 (NS) | 1.341 (NS) | 1.713 (NS) |
| N3 | 1.739 (<.05) | 5.451 (<.001) | 4.675 (<.001) |
| PA3 | 0.256 (NS) | 4.502 (<.001) | 4.808 (<.001) |
| PER | -0.406 (NS) | 2.958 (<.01) | 3.747 (<.001) |
| PSY | 0.709 (NS) | 1.920 (NS) | 0.397 (NS) |
| SUI | 0.024 (NS) | 1.921 (NS) | 2.618 (<.01) |
| (d.f.) | 177 | 111 | 190 |

# DISCUSSION

## A. Critical Discussion of Questionnaires

The questionnaires used in this study (KDS-1, KDS-2, KDS-3A) have received their greatest usefulness in the study of patients with affective illness, as well as functioning as a "screen" for psychiatric complaints. Both aspects fit well the scope of this study, in that affective disturbances are prominent but by no means the sole form of psychopathology in temporal lobe epilepsy.

The most important advantage of this technique of study of psychopathology is the freedom from observer bias which it allows. Inasmuch as the patient responds to the questions however he sees fit, without the inhibiting presence of the examiner or the unknown effects of his non-verbal judgments, and the computer scores the questionnaires following an objective program, there is no opportunity for the examiner to distort the transfer from patient complaint or difficulty to score on a symptom cluster scale. Of course, any technique that relies on the patient to determine his own degree of difficulty is subject to two problems: lack of honesty in completing the questionnaire, which is probably lack of confidence in the examiner, and the possible lack of sufficient cleverness in questionnaire design and analysis to reveal the difficulties the patient has described. The former problem has not been controlled for in this study, and would have required the coincident administration

of some other rating system known to be able to detect such reluctance. It might be added that the first problem in describing psychopathology is trusting the patient, so that his lack of forthrightness or confidence in completing a questionnaire is not so much a distressing technical consideration as it is another form of psychological difficulty to be detected and attended to. There is no place for tricks, games or deceit in the business of psychiatry.

That the questionnaires are indeed well-designed is revealed by the clarity with which they have outlined the psychiatric symptomatology of this group. Problems of hesitation, reservation and lack of confidence in the examiner are revealed in scales attempting to measure these items (PA, PA3). It would, of course, be more accurate to know the variation of each scale in a group of patients with the psychiatric diagnosis that names that scale, and to use this as a control in the comparison with the present study group. For technical reasons, this was not possible at the present time. However, it might be pointed out that the assignment of psychiatric diagnoses is itself an arbitrary collection of signs and symptoms (similar to those coded into the scales) which psychiatrists agree describe the entity to be named as the patient's problem. Thus the scales are a codification of common psychiatric practice, and more important, a quantification of psychiatric difficulty allowing comparison of varying degrees of severity of disturbance.

Thus the significance of the questionnaires as instruments used in the present study ought to be considered as follows.

1. They are free of observer bias.
2. They rely on the patient's ability to describe his problems.
3. They are quantified according to a defined and reproducible code for comparison of performance as it varies with other conditions.
4. They describe well affective psychopathology, a significant problem among temporal lobe epileptics and difficult to measure accurately.
5. They identify a series of seventeen scales which organize the responses and facilitate comparisons.

That the names for the scales ought not to be considered more specific than the items which comprise the scale has been mentioned, and the scales might as easily have been numbered from one to seventeen, except that the names make it easier to recall the contributing items.

It must be apparent from this analysis that, although far from perfect, the questionnaires used were well suited to the task of the study, and superior to the techniques of observation of psychopathology used in many of the studies reviewed above.

B. Relation of the Present Study to Existing Literature

This study is the first attempt to study affective psychopathology in temporal lobe epilepsy in an objective and quantitative way, and thus cannot be compared to existent literature on these bases. Because patients were not labeled "anxious" or "depressed" or "dysphoric" as they were in the studies reviewed above in Chapter Six, but instead were considered to have aspects of many different such entities in varying degrees, it is difficult to begin to compare the spectrum of psychopathology in the group studied with those previously reported.

If, as an arbitrary figure, we assume that a "Yes" response to enough items in each scale such that the score is at least 50% of the maximum score for that scale represents severe enough involvement to have been noted as such in previous studies, the data of Table D-1 emerge. Comparison of these data with previous studies is shown in Table D-2. A further measure of psychopathology is psychiatric hospitalization. 17 of 63, or 27%, of the present study had been hospitalized for psychiatric difficulty; in Stevens' study (4), 28 of 100 had been so hospitalized.

The lack of precision with which comparisons can be made reflects the design of the study, which was less to catalogue psychopathology than it was to describe problems and their relationships to measured parameters. There is no single, unitary picture that emerges from this study---no

Table D-1

Number of Patients With Half-Maximal (More Severe) Scores

| Scale | No. Pts. Severely Aff. | Scale | No. Pts. Severely Aff. |
|---|---|---|---|
| Anxiety | 9 | Chronic Anxiety | 32 |
| Depression | 20 | Chronic Depression | 32 |
| Psychosis | 8 | Chronic Psychopathy | 13 |
| Organicity | 17 | Personality Disorder | 21 |
| Neurosis | 19 | Chronic Neuroticism | 12 |
| Suicidal Ideation | 12 | Chronic Suicidal Ideation | 15 |
| Impulsivity | 21 | Chronic Impulsivity | 14 |
| Paranoia | 8 | Chronic Paranoia | 20 |
| Mania | 15 | | |

Table D-2

Comparison of Present Study with Existent Literature (Per Cent)

| Item | Present Study | Mulder & Daly(1) | Pond & Bidwell(2) | Currie et.al.(3) |
|---|---|---|---|---|
| Anxiety | 14 | 36 | -- | 19 |
| Depression | 32 | 16 | -- | 11 |
| Neurosis | 30 | -- | -- | -- |
| Psychosis | 13 | 4 | -- | 2 |
| Personality Disorder | 33 | -- | 51 | -- |
| N (all temporal lobe epileptics) | 63 | 100 | 39 | 666 |

thumbnail description which will characterize the psychopathology of temporal lobe epileptics. The process, as I have mentioned, is a heterogeneous one, and the causes of psychopathology are many and complex, different for each patient with the symptom of temporal lobe seizures. Table D-1 gives, not a catalogue, but a picture of the extent and severity of these psychiatric difficulties.

A number of relationships were examined in this study because of claims for their existence by other researchers. I could not confirm a relationship between higher seizure frequency and lower social class, as found by Pond and Bidwell (2). The populations studied are somewhat different, theirs being patients in general practice and these being patients of a university hospital clinic. Also, as mentioned, the precision of determination of social class in this study may not have been sufficient to reveal a relation to seizure frequency.

Flor-Henry has claimed that the laterality of EEG focus is related to the psychiatric manifestations (5). Although there was a suggestion that depression tended to be more severe in patients with a right temporal lobe EEG focus and psychosis more severe in patients with a left temporal EEG focus, I have mentioned that I did not consider the finding very significant. Flor-Henry could not present evidence that his findings were statistically significant, and in the study here the correlation was not of a very high degree. It should also be mentioned that, as reviewed in the chapter on

diagnosis and EEG (Chapter Four), the localization of temporal epileptic activity by EEG is not always precise or reproducible, many patients with recorded foci will show bilateral foci if appropriate techniques are applied, and the focus may shift from side to side with time. Experimental studies (Chapter Two) and surgical studies (Chapter Five) have similarly failed to establish a correlation between psychiatric difficulty and cerebral laterality.

The design of the study was not such as to confirm or refute the contention that temporal lobe epileptics are no different from other epileptics in their psychopathology, and this position must be adduced from the literature reviewed above. There were not sufficient patients with other forms of epilepsy in the clinic who could be matched with the patients with temporal lobe epilepsy in terms of seizure frequency and control, due to the quality of care received from Dr. Gallagher, Dr. Mattson and the others involved in their care, and the attempt to gather an appropriate control group had to be abandoned for this study.

Comparison of the results of this study with those of others who studied only temporal lobe epileptics with psychosis is imprecise, as certain relationships may only obtain when the psychopathology is of sufficient severity to warrant hospitalization and the diagnosis of "psychosis." It was not always possible to make such diagnoses in this group; none were hospitalized for psychiatric difficulties at the time they were studied; and the retrospective finding

of previous hospitalizations is much too inaccurate to permit comparison of only this subgroup with others' "psychotic" groups.

The findings of Slater and Beard (6) are not confirmed by this study. No relationship emerged between duration of epilepsy and severity of the scale for Psychosis. The present age and mean duration of epilepsy were sufficient in this study to reveal such a relation, according to their report, if one existed in these patients.

The claimed inverse relationship between psychosis and seizure frequency, reviewed in Section E of Chapter Six, was not found to hold for the patients of this study. It was not possible to evaluate the idea of "forced normalization" of the EEG as serial tracings were not done and no patient was actively psychotic at the time of testing. Brady's study of antisocial behavior in hospitalized temporal lobe epileptics (7) is not directly comparable to the present study, as these patients were not hospitalized and his "Disturbed Behavior Index" was not used. However, his finding that there was a trend, not statistically significant, for behavior to be worse in those with more frequent seizures, is related to the finding of this study that certain indices of psychopathology are more severe in those with more frequent seizures.

C. Contributions to the Existing Literature

It is apparent that the questionnaires used in this study can determine scales, the scores for which will vary with the frequency of seizures in temporal lobe epilepsy. It cannot be stated whether they distinguish patients with temporal lobe epilepsy from those with other forms of epilepsy. The scales will distinguish with a high degree of significance the degree of psychopathology in patients with temporal lobe epilepsy from that of normal people and that of patients treated for primary affective illness and in remission. The magnitude of this difference reveals the need for careful studies of treatment of psychopathology in temporal lobe epileptics, and the results provide a baseline for such study in that they describe what that psychopathology is in this group of patients. As only one example, the results of folate supplementation might have been more clear if Reynolds and his group had attempted an observer-blind, quantitative measurement of psychological status such as is possible with the techniques of this study.

A number of statements can be made about the determinants of psychopathology in temporal lobe epilepsy as a result of this study.

The increase in score for the scales Depression (D), Organicity (O), and Suicidal Ideation (SU) with seizure frequency is related to the overlap of items which determine these scales, as described in Table M-1 above. Thus it cannot be stated with certainty which of these scales,

or if all of them, vary with seizure frequency. Similarly, Chronic Anxiety (A3) and Chronic Suicidal Ideation (SUI) share several items in the determination of each respective scale, as do Impulsivity (I) and Mania (M). Anxiety (A) shares relatively few items with any other scale. Thus there are four groups of symptom cluster scales which vary with seizure frequency that are relatively independent of each other. Of course, it cannot be said whether the increase in seizures is responsible for the increase in severity of psychiatric difficulty, or whether the increase in psychiatric difficulty precipitated an exacerbation of seizure control. (See also below, Suggestions for Further Research.)

Social class and duration of epilepsy seem to operate in opposite directions in the determination of the extent of suicidal thinking. Social class also seems to exacerbate other psychiatric difficulties (Anxiety, Psychosis, Neurosis, Chronic Anxiety), although the lack of precision in determination of social class requires qualification of this statement. Duration of epilepsy also seems to temper the entity named Chronic Impulsivity, which shares several items with Chronic Suicidal Ideation but which may be a related process in the patient's mind.

The presence of a history of traumatic etiology seems to imply more severe psychopathology than if the etiology is unknown. Perseveration, a measure of mental functioning, is loosely related to a traumatic etiology, but more closely related to a lower seizure frequency.

Although as a group the patients demonstrate impaired mental functioning (Table R-2, Part B), it is not severe and not disproportionate to the level of intelligence necessary to understand the questionnaires. The disturbance of mental status was not related to any of the parameters measured.

Electroencephalographic foci, psychiatric history and social functioning have indirect and interacting roles in the manifestation of psychopathology as measured here. In addition, social functioning is related on the one hand to seizure frequency and on the other to psychiatric hospitalization.

D. Reconsideration of Hypotheses

In the Introduction to this paper, I outlined three theoretical explanations of the psychopathology of temporal lobe epilepsy, one a medical model, one a social model, and one a mixed (social, psychological and medical) model. The results of this study make it difficult to accept a solely social model, as the relationship to seizure frequency is too impressive to be ignored. Similarly, an explanation based only on epileptic activity is difficult to reconcile with the findings of significant variation with other parameters, and the incomplete variation (only eight of the seventeen scales) with seizures. Clearly, psychopathology cannot be said to be determined by either a patient's social status or by the presence of temporal lobe seizures alone. Not unexpectedly, the causation is a complex one, and it is probably true that in each patient many factors operate to a varying degree to determine the nature and the severity of psychopathology. The psychopathology of epilepsy, like epilepsy itself, is an entity of heterogeneous nature, and awaits further understanding before causation and determining relationships can be stated with a modest certainty. I do not consider the mixed model of causation satisfactory, and further research with techniques similar to those of this study might help elucidate the relative importance of those factors included in the hypothesis of a mixed social, medical and psychological causation.

E. Implications for Further Research

Several potential lines of investigation are possible as a result of this study, and which might help clear up some of the ambiguities in our understanding of this difficult and important problem. An important element, although one in which the techniques of this study would not be very useful, would be the elucidation in the laboratory of the correlations between clinical phenomena and anatomical, physiological and electrical phenomena, aiding immensely in the separation of the epileptic process into homogeneous entities which could be studied in greater detail. Other possible investigations using the methods of this study include:

1. Comparison of matched groups of temporal lobe epileptics and non-temporal lobe epileptics would help to identify the contribution of temporal lobe seizures to observed psychopathology. Propagation of generalized discharges through temporal lobe discharges would have to qualify such a result.
2. More precise scales for social class and seizure frequency, as well as the use of serum anticonvulsant concentrations instead of drug dosage, would make make comparison of these parameters more accurate.
3. Delineation of the variation of the scales used in this study in groups of psychiatric patients

having similar diagnosis would aid in interpretation of the meaning of scores on these scales.

4. Examination of the variation of each item on the questionnaires with seizure frequency would identify those problems most closely related to the activity of epilepsy.
5. Comparison of responses for each item to those of properly controlled groups of psychiatric patients would help identify those problems which are most important for patients with epilepsy.
6. As a result of #4 and #5, a special questionnaire could be constructed to focus more specifically on the psychopathology of epilepsy.
7. Using either the methods of this study or the method implied in #6, longitudinal studies of the impact of many parameters, including social, psychological, medical and therapeutic ones, could be evaluated more precisely.

It is apparent that a great deal of work remains to be done in order to obtain an understanding of the clinical problem of psychopathology in epilepsy and in epileptics with temporal lobe seizures. I shall be happy if this study can be an aid to this work.

References for Discussion

1. Mulder, D.W., Daly, D. J.A.M.A. 130:173 (1952)

2. Pond, D.A., Bidwell, B.M. Epilepsia 1:285 (1960)

3. Currie, S., Heathfield, K.W., Henson, R.A., Scott, D.F. Brain 94:173 (1971)

4. Stevens, J.R. Arch. Gen. Psychiat. 14:461 (1966)

5. Flor-Henry, P. Epilepsia 10:363 (1969)

6. Slater, E., Beard, A.W., Glithero, E. Brit. J. Psychiat. 109:95 (1963)

7. Brady, J.P. J. Nerv. Ment. Dis. 138:468 (1964)

## CONCLUSIONS

1. Sixty-three patients with temporal lobe epilepsy were given computer-scored questionnaires and the results correlated with social, psychological and neurological information.
2. Temporal lobe epileptics have significantly more psychopathology than either normal people or patients with primary affective illness in remission and under treatment. The differences are not confined to a single or several scales for measurement of that psychopathology.
3. Scales composed of well-defined items have been shown to be related to the frequency of temporal lobe seizures in the severity of disturbance they measure.
4. Social class, psychiatric history, etiology of seizures, and duration of epilepsy modified the response of temporal lobe epileptics to items measured in the scales.
5. No evidence was found to support a relationship between psychosis as measured and seizure frequency or seizure duration.
6. The role of the right temporal lobe in depression and the left temporal lobe in psychosis is suggested.
7. The literature is extensively reviewed, and the psychopathology of temporal lobe epilepsy is considered to be of a mixed causation, and further investigation is suggested to clarify the issues.

# KDS-1™ *

0. 2 PENCIL

DO NOT MARK IN SHADED AREA

| T NAME | DATE | TIME OF DAY |
|---|---|---|
| | | |

DATE: FEB MAR APR MAY JUN JUL AUG SEP OCT DEC 71 72 73 74 75

PATIENT: 0 1 2 3 4 5 6 7 8 9

FACILITY CODE: 1 2 3 4 5 6 7 8 9

SEX OF PATIENT: MALE FEMALE

MARK ONE: AM PM EVENING NIGHT

NSWER EVERY ITEM BY SHADING IN THE YES OR NO COLUMN. EXAMPLE: YES NO

| | | |
|---|---|---|
| NO | I FEEL DOWNHEARTED AND BLUE | |
| NO | I FEEL LIKE GOING OUT AND SPENDING MONEY | |
| NO | I SIMPLY DON'T HAVE THE ENERGY TO DO THINGS THE WAY I USED TO | |
| NO | IF I WATCH VERY CAREFULLY, I CAN SEE OR HEAR THINGS OTHER PEOPLE CAN'T | |
| NO | I AM SCARED OF GOING TO NEW PLACES OR MEETING NEW PEOPLE | |
| NO | I AM ENJOYING MY FOOD | |
| NO | WHEN I MEET PEOPLE I FEEL I HAVE NOTHING TO SAY | |
| NO | I FIND THAT MY THINKING HAS SLOWED DOWN RECENTLY | |
| NO | I FEEL RESTLESS AND I CANNOT KEEP STILL | |
| NO | I HAVE RECENTLY STARTED TO AVOID RIDING TRAINS, ELEVATORS, BRIDGES OR BEING IN ENCLOSED AREAS OR HIGH PLACES | |
| NO | I FIND IT INCREASINGLY DIFFICULT TO MAKE DECISIONS | |
| NO | NOISES ALL SEEM TO BE LOUDER TO ME THAN THEY WERE BEFORE. IT'S AS IF SOMEONE HAD TURNED UP THE VOLUME | |
| NO | I FEEL IRRITABLE WHEN PEOPLE INTERFERE WITH MY PLANS | |
| NO | I AM DREAMING NOW MORE THAN I USUALLY DO | |
| NO | I FEEL THAT THINGS ARE LOOKING UP AND THAT I CAN ACCOMPLISH GREAT THINGS | |
| NO | I FEEL THAT OTHERS WOULD BE BETTER OFF IF I WERE DEAD | |
| NO | I HAVE A FEELING THAT PEOPLE ARE AGAINST ME | |
| NO | MY THOUGHTS ARE RACING SO FAST THAT IT FRIGHTENS ME | |
| NO | I AM HAVING VERY STRANGE EXPERIENCES | |
| NO | I NOW HAVE MORE SEXUAL DESIRES THAN I HAVE HAD FOR SOME TIME | |
| NO | WHEN I AM TALKING TO SOMEBODY, THE SLIGHTEST THING DISTRACTS ME, EVEN IF A PERSON ONLY CROSSES HIS LEGS | |
| YES NO | I RECENTLY HAVE HAD MORE TROUBLE EXPRESSING MY THOUGHTS OR SAYING WHAT I WANT TO SAY | |
| YES NO | I FEEL LIKE TALKING WITH PEOPLE, EVEN IF IT'S NECESSARY TO PHONE THEM LONG DISTANCE | |
| YES NO | I HAVE BEEN WAKING UP EARLIER THAN I NEED TO | |
| YES NO | I CANNOT STOP MYSELF FROM CHECKING THINGS OVER AND OVER | |
| YES NO | CERTAIN THOUGHTS COME IN MY MIND AND I CAN'T KEEP THEM OUT | |
| YES NO | SOMETIMES I GET FRIGHTENED BECAUSE EVERYTHING SEEMS FAR AWAY | |
| YES NO | WHEN I AM WITH CERTAIN PEOPLE, VERY PECULIAR THINGS BEGIN TO HAPPEN | |
| YES NO | I AM SLEEPING MORE THAN I USED TO | |
| YES NO | NOWADAYS I FEEL WORSE IN THE MORNING | |
| YES NO | MY MEMORY IS NOT AS SHARP AS IT USED TO BE | |
| YES NO | I SUSPECT SOMEONE IS TRYING TO MAKE ME DO THINGS | |
| YES NO | LATELY I FIND MYSELF LOSING MY TEMPER | |
| YES NO | NOWADAYS WHEN I GET ANXIOUS, MY HEART STARTS TO BEAT FAST | |
| YES NO | AT TIMES I AM NOT SURE WHETHER I AM AWAKE OR DREAMING | |
| YES NO | MY EVENINGS AND NIGHTS ARE MY BEST TIMES | |
| YES NO | I AM UP ALL NIGHT AND SLEEP DURING THE DAY | |
| YES NO | I AM NAPPING MORE THAN I USED TO | |
| YES NO | LAST NIGHT I SLEPT SOUNDLY | |
| YES NO | LAST NIGHT I HAD TROUBLE GETTING TO SLEEP | |
| YES NO | LAST NIGHT I WOKE UP MORE THAN ONCE | |



IBM M 63741

# KDS-2™*



NO. 2 PENCIL. PLEASE ANSWER EVERY ITEM BY SHADING EITHER YES OR NO

DO NOT MARK IN SHADED AREA

| T NAME | DATE | TIME OF DAY |
|---|---|---|
| | | |

DATE

JAN FEB MAR APR MAY JUN JUL AUG SEP OCT
NOV DEC 71 72 73 74 75
1 2 3 4 5 6 7 8 9

PATIENT

0 1 2 3 4 5 6 7 8 9

FACILITY CODE

1 2 3 4 5 6 7 8 9

SEX OF PATIENT: MALE FEMALE

MARK ONE: AM PM EVENING NIGHT

IF ANYBODY HAS PRESCRIBED MEDICATION FOR YOU DURING THE LAST WEEK, PLEASE MAKE SURE YOU REPORT IT.

| C'OU HAVE ANY OF THE FOLLOWING YPTOMS? | | | IF YES, DOES IT MAKE YOU VERY UNCOMFORTABLE? | |
|---|---|---|---|---|
| | :NO | DROWSINESS | YES | NO |
| | :NO | STIFFNESS OF MUSCLES IN LEGS OR ARMS | YES | NO |
| | :NO | VAGINAL DISCHARGE | YES | NO |
| | :NO | SHORTNESS OF BREATH | YES | NO |
| | :NO | BLEEDING GUMS | YES | NO |
| | :NO | TREMORS OR "SHAKES" | YES | NO |
| | :NO | DIFFICULTY IN SWALLOWING | YES | NO |
| | :NO | SPASMS OF NECK OR TONGUE | YES | NO |
| | :NO | RESTLESSNESS, INABILITY TO BE STILL | YES | NO |
| | :NO | ITCHING | YES | NO |
| | :NO | POOR MEMORY | YES | NO |
| | :NO | DIZZINESS | YES | NO |
| | :NO | CLUMSINESS | YES | NO |
| | :NO | COLD HANDS OR FEET | YES | NO |
| | :NO | NUMBNESS OR TINGLING OF LEGS OR ARMS | YES | NO |
| | :NO | FAINTING SPELLS | YES | NO |
| | :NO | LOSS OF HAIR | YES | NO |
| | :NO | BLURRED VISION | YES | NO |
| | :NO | HEADACHE | YES | NO |
| | :NO | NERVOUSNESS | YES | NO |
| | :NO | NAUSEA OR VOMITING | YES | NO |
| | :NO | DIFFICULTY GETTING OR MAINTAINING AN ERECTION | YES | NO |
| | :NO | DIFFICULTY IN EJACULATING | YES | NO |
| | :NO | DIARRHEA | YES | NO |
| | :NO | CONSTIPATION | YES | NO |
| | :NO | DRY MOUTH AND THROAT | YES | NO |
| | :NO | INCREASED SALIVATION | YES | NO |
| | :NO | NASAL STUFFINESS | YES | NO |
| | :NO | NIGHTMARES | YES | NO |
| | :NO | CHILLS | YES | NO |
| | :NO | IRREGULAR HEART BEAT | YES | NO |
| | :NO | COUGH | YES | NO |

| DO YOU HAVE ANY OF THE FOLLOWING SYMPTOMS? | | | IF YES, DOES IT MAKE YOU VERY UNCOMFORTABLE? | |
|---|---|---|---|---|
| YES | NO | DECREASED APPETITE | YES | NO |
| YES | NO | SKIN RASH | YES | NO |
| YES | NO | BLOODY URINE | YES | NO |
| YES | NO | JAUNDICE (YELLOW DISCOLORATION OF SKIN OR EYES) | YES | NO |
| YES | NO | FEVER | YES | NO |
| YES | NO | DIFFICULTY STARTING URINATION | YES | NO |
| YES | NO | FREQUENT URGE TO URINATE | YES | NO |
| YES | NO | INABILITY TO URINATE | YES | NO |
| YES | NO | LACK OF ENERGY | YES | NO |
| YES | NO | IRREGULARITY OR ABSENCE OF MENSTRUAL PERIOD | YES | NO |
| YES | NO | NOSEBLEED | YES | NO |
| YES | NO | WEAKNESS IN LEGS OR ARMS | YES | NO |
| YES | NO | DARK URINE | YES | NO |
| YES | NO | PAINS OR CRAMPS IN THE ABDOMEN | YES | NO |
| YES | NO | SWELLING OF BREASTS | YES | NO |
| YES | NO | HISSING NOISES IN EARS | YES | NO |
| YES | NO | FLUID DISCHARGE FROM BREAST | YES | NO |
| YES | NO | DIFFICULTY MAINTAINING SEXUAL EXCITEMENT | YES | NO |
| YES | NO | DIFFICULTY IN REACHING ORGASM | YES | NO |
| YES | NO | INCREASED SWEATING | YES | NO |
| YES | NO | EDEMA (SWELLING) OF LEGS | YES | NO |
| YES | NO | UNSTEADY GAIT (POOR BALANCE) | YES | NO |
| YES | NO | TEARING EYES | YES | NO |
| YES | NO | SLURRED SPEECH | YES | NO |
| YES | NO | PAINFUL SORES INSIDE MOUTH | YES | NO |
| YES | NO | RAPID OR POUNDING HEART BEAT | YES | NO |
| YES | NO | INCREASED APPETITE | YES | NO |
| YES | NO | INCREASED THIRST | YES | NO |
| YES | NO | CRAMPS IN LEGS OR ARMS | YES | NO |
| YES | NO | CHEST PAIN | YES | NO |
| YES | NO | BACK PAIN | YES | NO |
| YES | NO | PAINFUL INTERCOURSE | YES | NO |

*T KDS Form was developed by David J. Kupfer, M.D. & Thomas P. Detre, M.D.

IBM M63482

K S-3A-71

# KDS™-3A*

208

NUMBER 2 PENCIL

DO NOT MARK IN SHADED AREA

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0 1 2 3 4 5 6 7 8 9

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0 1 2 3 4 5 6 7 8 9

| I WANT TO ASK YOU ABOUT THE WAY YOU USUALLY FEEL OR ACT..... | MOST OF YOUR LIFE | | ONLY RECENTLY | |
|---|---|---|---|---|
| DO YOUR MUSCLES TIGHTEN UP WHEN YOU BECOME ANXIOUS? | YES | NO | YES | NO |
| DO YOU LIKE BEING IN THE MIDST OF EXCITEMENT? | YES | NO | YES | NO |
| DO YOU OFTEN FEEL YOU ARE POSING OR PRETENDING? | YES | NO | YES | NO |
| DO YOU FEEL MORE RELAXED INDOORS? | YES | NO | YES | NO |
| DO YOU FEAR BEING LEFT ALONE? | YES | NO | YES | NO |
| DO YOU FEEL PANICKY IN CROWDS? | YES | NO | YES | NO |
| DO YOU OFTEN HAVE PERIODS DURING WHICH EVERYTHING AND EVERYBODY SEEMS UNREAL AND FAR AWAY? | YES | NO | YES | NO |
| DO YOU TEND TO BE LATE FOR APPOINTMENTS? | YES | NO | YES | NO |
| DO YOU OFTEN ASKED YOURSELF "HAVE I DONE THE RIGHT THING?" | YES | NO | YES | NO |
| DO YOU FEEL COMPELLED TO COUNT THINGS? | YES | NO | YES | NO |
| DO YOU STOP AND THINK BEFORE DOING THE SMALLEST THING? | YES | NO | YES | NO |
| DO YOU LIKE MAKING QUICK DECISIONS? | YES | NO | YES | NO |
| DO YOU TEND TO LOSE YOUR TEMPER WHEN YOU DON'T GET YOUR WAY? | YES | NO | YES | NO |
| DO YOU OFTEN GET A HEADACHE WHEN YOU FEEL TENSE? | YES | NO | YES | NO |
| DO YOU OFTEN WORRY THAT SOMETHING BAD IS GOING TO HAPPEN? | YES | NO | YES | NO |
| DO YOU GET ANXIOUS WHEN YOU HAVE TO ASK SOMEONE FOR A FAVOR? | YES | NO | YES | NO |
| DO YOU GET ANXIOUS WHEN YOU HAVE TO MEET NEW PEOPLE OR GO TO A NEW PLACE? | YES | NO | YES | NO |
| DO YOU USUALLY TRY TO BE THE CENTER OF ATTENTION OR THE LIFE OF THE PARTY? | YES | NO | YES | NO |
| ARE YOU CONCERNED THAT PEOPLE THINK YOU ARE UNATTRACTIVE? | YES | NO | YES | NO |
| DO YOU BECOME EASILY ENTHUSIASTIC AND JUST AS EASILY DISAPPOINTED ABOUT PEOPLE? | YES | NO | YES | NO |
| DO YOU FEEL BAD FOR A LONG TIME WHEN SOMEBODY CRITICIZES YOU? | YES | NO | YES | NO |
| DO YOU FEEL THAT YOUR LIFE IS IN A RUT AND THAT YOU ARE UNABLE TO CHANGE IT? | YES | NO | YES | NO |
| DO YOU FIND THAT MOST PEOPLE DEEP DOWN ARE FULL OF HATRED? | YES | NO | YES | NO |
| DO YOU BECOME TIRED AND EXHAUSTED EASILY? | YES | NO | YES | NO |
| DO YOU THINK PEOPLE FIND YOU SEXUALLY ATTRACTIVE? | YES | NO | YES | NO |
| DO YOU GET INTO FIGHTS WITH PEOPLE YOU LOVE? | YES | NO | YES | NO |
| DO YOU OFTEN FEEL ANNOYED WITH PEOPLE WHO ARE NOT ORDERLY? | YES | NO | YES | NO |
| CAN YOU EASILY BE CHEERED UP EVEN WHEN YOU GET VERY UPSET? | YES | NO | YES | NO |
| DO YOU OFTEN WORRY ABOUT YOUR PHYSICAL HEALTH? | YES | NO | YES | NO |
| ARE YOU ALWAYS AFRAID OF CATCHING SOMEBODY'S ILLNESS OR BEING CONTAMINATED? | YES | NO | YES | NO |
| DO YOU OFTEN THINK ABOUT THINGS THAT HAPPENED WHEN YOU WERE A CHILD? | YES | NO | YES | NO |
| ARE YOU OFTEN CONCERNED THAT SOMEHOW YOU ARE GOING TO LOSE CONTROL AND HURT YOURSELF OR SOMEBODY ELSE? | YES | NO | YES | NO |
| DO YOU TEND TO GET INTO ACCIDENTS? | YES | NO | YES | NO |
| WHEN THINGS GO WRONG IN YOUR LIFE, DO YOU THINK OF IT AS SOME SORT OF PUNISHMENT? | YES | NO | YES | NO |
| DO YOU FIND IT DIFFICULT TO GET CLOSE TO PEOPLE? | YES | NO | YES | NO |
| DO YOU GET BORED WITH ALMOST EVERYTHING YOU DO? | YES | NO | YES | NO |

| IN THE YEARS, WITHOUT ANY APPARENT REASON YOU SOMETIMES FELT..... | | IF YES, FOR HOW LONG? (INDICATE WHETHER DAYS, WEEKS, OR MONTHS) HOURS | DAYS | WEEKS | MONTHS |
|---|---|---|---|---|---|
| YES NO | VERY HAPPY ..... | (----) | (----) | (----) | (----) |
| YES NO | VERY DEPRESSED ..... | (----) | (----) | (----) | (----) |
| YES NO | SOMETIMES HAPPY, SOMETIMES DEPRESSED ..... | (----) | (----) | (----) | (----) |

| | | | | |
|---|---|---|---|---|
| WHEN SOMETHING UPSETS YOU, DO YOU SLEEP.....MORE (----) | LESS (----) | THE SAME (----) | | |
| WHEN SOMETHING UPSETS YOU, DO YOU EAT .....MORE (----) | LESS (----) | THE SAME (----) | | |
| WHEN SOMETHING UPSETS YOU, DOES YOUR SEXUAL INTEREST...INCREASE (----) | | DECREASE (----) | REMAIN UNCHANGED | (----) |

KDS Form developed by David J. Kupfer, M.D. & Thomas P. Detre, M.D., Yale University School of Medicine.



IBM M63220

# KDS-5™*
## MENTAL STATUS



USE NUMBER 2 PENCIL — DO NOT MARK IN SHADED AREAS

NAME OF PATIENT | DATE | TIME | RATER'S INITIALS

DATE: FEB MAR APR MAY JUN JUL AUG SEP OCT / DEC 71 72 73 74 75 / 1 2 3 4 5 6 7 8 9

FACILITY CODE: 1 2 3 4 5 6 7 8 9

RATER CODE: 1 2 3 4 5 6 7 8 9

PATIENT: 0 1 2 3 4 5 6 7 8 9

AGE OF PATIENT: 0 1 2 3 4 5 6 7 8 9

SEX OF PATIENT: MALE ( ) FEMALE ( )

RELIGION OF PATIENT: PROTESTANT ( ) CATHOLIC ( ) JEWISH ( ) OTHER ( ) NONE ( )

MARITAL STATUS: SING ( ) MAR ( ) SEP ( ) DIV ( ) DIV, REMAR ( ) WID, REMAR ( ) WID ( )

MIC BACKGROUND: CAUCASIAN (WHITE) ( ) NEGRO (BLACK) ( ) PUERTO RICAN ( ) ORIENTAL ( ) INDIAN (AMERICAN) ( ) OTHER ( )

MARK ONE: AM ( ) PM ( ) EVENING ( ) NIGHT ( )

**APPEARANCE** (MARK ALL THAT APPLY)

UNUSUALLY ATTRACTIVE ( ) UNUSUALLY UNATTRACTIVE ( ) MARKED FLUSHING ( )
VISIBLE PHYSICAL DEFORMITY PRESENT ( ) MARKED FACIAL PALLOR ( )

**E AND GROOMING:**

ACCEPTABLE (APPROPRIATE) ( ) CARELESS (SLOPPY) ( ) CANNOT BE ASSESSED (BEDRIDDEN) ( )
METICULOUS (FUSSY) ( ) BIZARRE ( )

**IT:** ABOUT RIGHT ( ) APPEARS GROSSLY OVERWEIGHT ( ) APPEARS GROSSLY UNDERWEIGHT ( )

**OR ACTIVITY AND PATTERNING:**

DEN ( ) GRACEFUL ( ) UNUSUAL GAIT ( ) MOVES OR THRASHES AROUND RESTLESSLY ( )
NED TO WHEELCHAIR ( ) CLUMSY ( ) POVERTY OF ASSOCIATED MOVEMENTS ON AMBULATION ( )
AL ONLY WITH ASSISTANCE ( ) CHOREIFORM MOVEMENTS ( ) KEEPS STILL MOST OF THE TIME ( )
TICS ( ) HANDS IN CONSTANT MOTION ( ) TREMOR ( )

**EECH:**

AR D REGIONAL DIALECT ( ) EASILY UNDERSTANDABLE ( ) LISP ( ) WORD-FINDING DIFFICULTY ( )
ATI TONGUE OTHER THAN ENGLISH ( ) MONOTONOUS ( ) STUTTER ( ) MUTE ( )
HALTING ( ) SLURRED ( ) AFFECTED ( )

**NSORY SYSTEMS:** VISION: MARKED IMPAIRMENT ( ) BLIND ( ) HEARING: MARKED IMPAIRMENT ( ) DEAF ( )

**ATTITUDE DISPLAYED DURING INTERVIEW** (MARK ALL THAT APPLY)

FRIENDLY ( ) EVASIVE ( ) INGRATIATING ( )
FEARFUL ( ) SARCASTIC ( ) UNRESPONSIVE ( )

**PATIENT IS UNRESPONSIVE, BY-PASS SECTION "C" AND CONTINUE WITH SECTION "D"**

**ASK THE PATIENT:**

WHAT IS THE NAME OF THIS PLACE? _____ CORRECT ( ) INCORRECT ( )

WHAT IS TODAY'S DATE? MONTH _____ DAY _____ YEAR _____ CORRECT ( ) INCORRECT ( )

WHAT ARE THE NAMES OF THE PAST THREE PRESIDENTS OF THE U. S.? CORRECT ( ) INCORRECT ( )

EN ASK PATIENT TO COUNT BY ONES TO 23. ABLE TO DO IT? YES NO

EXT ASK THE FOLLOWING TWO QUESTIONS....

WHAT ARE THE COLORS IN THE AMERICAN FLAG? RED BLUE WHITE CORRECT ( ) INCORRECT ( )

WHAT IS A THERMOMETER? INSTRUMENT (THING, ETC.) FOR MEASURING TEMPERATURE CORRECT ( ) INCORRECT ( )

OW ASK PATIENT TO SUBTRACT 7 FROM 100 (CONTINUE TO 51) (SERIAL SEVENS)

NUMBER OF MISTAKES: 1 2 3 4 5 PERSEVERATION? YES NO

**PATIENT HAS GIVEN INCORRECT ANSWERS TO ANY OF THE ABOVE QUESTIONS IN SECTION "C", OR MADE MORE THAN ONE MISTAKE IN SERIAL SEVENS, ADMINISTER SECTION "D".**

MARK LAST FOUR DIGITS OF ABOVE RED NUMBER

0 1 2 3 4 5 6 7 8 9
0 1 2 3 4 5 6 7 8 9
0 1 2 3 4 5 6 7 8 9
0 1 2 3 4 5 6 7 8 9

# KDS-5™* MENTAL STATUS



NEXT, ASK THE FOLLOWING TWO QUESTIONS...

HOW FAR IS IT FROM LOS ANGELES TO NEW YORK? ANYWHERE FROM 2000 TO 3000 MILES CORRECT (=====) INCORRECT (=====)

NAME THREE COUNTRIES IN THE MIDDLE EAST: ISRAEL–EGYPT–JORDAN–SYRIA–IRAQ–LEBANON–ETC. CORRECT (=====) INCORRECT (=====)

NOW, ASK PATIENT TO "CONTINUE COUNTING FROM WHERE YOU LEFT OFF, STARTING WITH THE NEXT NUMBER, UP TO 31"

ABLE TO COMPLETE THE TASK? :YES: :NO:

D.

IS COLLATERAL INFORMATION AVAILABLE? :YES: :NO:

IF NO, WILL COLLATERAL INFORMATION BE AVAILABLE AT SOME FUTURE TIME? :YES: :NO:

IF YES, SPECIFY INFORMANT _____, AND ADMINISTER THE FOLLOWING QUESTIONS....

CHANGES IN PATIENT'S PERFORMANCE AND HABITS DURING THE 72 HOURS PRIOR TO THIS EXAMINATION. (MARK ALL THAT APPLY)

(=====) INABILITY TO PERFORM HOUSEHOLD TASKS

(=====) INABILITY TO REMEMBER SHORT LISTS OF ITEMS (EXAMPLE, AS IN SHOPPING)

(=====) INCREASINGLY STUBBORN AND UNCOOPERATIVE

(=====) LACK OF REGARD FOR FEELINGS OF OTHERS

(=====) INCREASED IRRITABILITY

(=====) INAPPROPRIATE SEXUAL BEHAVIOR

(=====) DIMINISHED INITIATIVE OR GROWING APATHY

(=====) PURPOSELESS HYPERACTIVITY

EATING (=====) EATS WITHOUT HELP
(=====) NEEDS SOME HELP
(=====) NEEDS HELP AT ALL TIMES

DRESSING (=====) DRESSES WITHOUT HELP
(=====) NEEDS SOME HELP
(=====) NEEDS HELP AT ALL TIMES

SPHINCTER CONTROL (=====) SATISFACTORY
(=====) WETS BED OCCASIONALLY
(=====) DEFECATES IN BED

**FOR DETAILED HISTORY RECORD, ADMINISTER KDS-4F TO THE NEAREST RELATIVE OR ACQUAINTANCE**

**E. RESPONSES SHOULD BE MARKED ON THE BASIS OF PERSONAL OBSERVATION**

:YES: :NO: RESPONDS ONLY TO STRONG OR PAINFUL STIMULI

:YES: :NO: LACK OF SPONTANEOUS FACIAL OR OTHER NONVERBAL EXPRESSION

:YES: :NO: LACK OF EMOTIONAL EXPRESSION OR RESPONSE EVEN WHEN DIRECTLY APPROACHED OR STIMULATED

:YES: :NO: UNABLE TO FOLLOW EVEN SIMPLE INSTRUCTIONS

:YES: :NO: EXPRESSES EXAGGERATED OPINION OF SELF, BELIEVES HE IS EXCEPTIONALLY CAPABLE

:YES: :NO: EXPRESSES FEELINGS OF SADNESS, WORTHLESSNESS, FAILURE, HOPELESSNESS, REMORSE, GUILT OR LOSS

:YES: :NO: OBSERVABLE MANIFESTATIONS OF ANXIETY OR PANIC

:YES: :NO: VERBAL EXPRESSION OF OVERT HOSTILITY

:YES: :NO: PHYSICALLY THREATENING BEHAVIOR

:YES: :NO: DECREASED MOTOR ACTIVITY

:YES: :NO: INCREASED MOTOR ACTIVITY

:YES: :NO: DISORGANIZED SPEECH (DISCONNECTED OR BIZARRE)

:YES: :NO: UNUSUAL OR BIZARRE EXPRESSIVE BEHAVIOR (SUCH AS MIMIC, GESTURES, POSTURE)

:YES: :NO: SUSPICIOUS AND DISTRUSTFUL

:YES: :NO: CONCERNED WITH SUICIDE

:YES: :NO: OBVIOUSLY DELUSIONAL

:YES: :NO: PRESSURED SPEECH

:YES: :NO: OVERWHELMED WITH HOMOSEXUAL FEARS

:YES: :NO: DROWSY, INATTENTIVE

:YES: :NO: HYPERALERT, EASILY DISTRACTED

:YES: :NO: INTERMITTENTLY ALERT AND DROWSY

:YES: :NO: UNABLE TO GIVE A CHRONOLOGICAL ACCOUNT OF HIS ACTIVITIES

:YES: :NO: DISTINCT MEMORY IMPAIRMENT

:YES: :NO: LITTLE OR NO SPONTANEOUS SPEECH

:YES: :NO: PREOCCUPIED WITH PHYSICAL HEALTH

:YES: :NO: AFRAID OF SPECIFIC OBJECTS OR SITUATIONS (SUCH AS ELEVATORS, HEIGHT, CLOSED ROOMS, OR KNIVES)

:YES: :NO: REPEATS CERTAIN ACTS OR THINKS ABOUT THINGS OVER AND OVER AGAIN

:YES: :NO: CONSTANTLY SKIPS FROM ONE TOPIC TO ANOTHER

:YES: :NO: AVOIDS EYE CONTACT

:YES: :NO: MOOD FLUCTUATES BETWEEN ELATION AND SADNESS

:YES: :NO: ACTS LIKE A "PRACTICAL JOKER"

:YES: :NO: OVERTLY SEDUCTIVE (VERBALIZATION OR MANIFEST BEHAVIOR)

:YES: :NO: BEHAVIOR STRONGLY SUGGESTIVE OF HALLUCINATIONS

:YES: :NO: HALLUCINATORY EXPERIENCE DESCRIBED BY PATIENT

IF YES, MARK ALL THAT APPLY:

VISUAL (=====) GUSTATORY (=====)

TACTILE (=====) AUDITORY, IMPERATIVE (=====)

OLFACTORY (=====) AUDITORY, NON-IMPERATIVE (=====)

SIGNATURE(S): _____

MARK LAST FOUR DIGITS OF ABOVE RED NUMBER

:0: :1: :2: :3: :4: :5: :6: :7: :8: :9:

:0: :1: :2: :3: :4: :5: :6: :7: :8: :9:

:0: :1: :2: :3: :4: :5: :6: :7: :8: :9:

:0: :1: :2: :3: :4: :5: :6: :7: :8: :9:

Appendix 2

1. Anxiety (A)--Total 48 points

| Weight | Item |
|---|---|
| 2 | I feel restless and I cannot keep still. |
| 2 | I am dreaming now more than I usually do. |
| 3 | Nowadays, when I gen anxious my heart starts to beat fast. |
| 2 | Last night I had trouble getting to sleep. |
| 2 | Last night I woke up more than once. |
| | (Do you have any of the following symptoms?) |
| 2 | Stiffness of muscles in legs or arms |
| 3 | Shortness of breath |
| 3 | Tremors or "shakes" |
| 2 | Difficulty in swallowing |
| 2 | Restlessness, inability to keep still |
| 1 | Dizziness |
| 1 | Fainting spells |
| 2 | Headache |
| 3 | Nervousness |
| 1 | Nausea or vomiting |
| 2 | Diarrhea |
| 1 | Dry mouth or throat |
| 3 | Nightmares |
| 2 | Irregular heart beat |
| 2 | Frequent urge to urinate |
| 1 | Pains or cramps in the abdomen |
| 2 | Increasing sweating |
| 1 | Unsteady gait (poor balance) |
| 2 | Rapid or pounding heart beat |
| 1 | Chest pain |

2. Depression (D)--Total 55 points

| Weight | Item |
|---|---|
| 3 | I feel downhearted and blue. |
| 2 | I simply don't have the energy to do things the way I used to. |
| 3 | I am enjoying my food. (Score only if "No.") |
| 3 | When I meet people I feel I have nothing to say. |
| 3 | I find that my thinking has slowed down recently. |
| 3 | I find it increasingly difficult to make decisions. |
| 1 | I am dreaming more now than I usually do. (Score if "No.") |
| 1 | I feel that things are looking up and I can accomplish great things. (Score if "No.") |
| 3 | I feel that others would be better off if I were dead. |
| 1 | I now have more sexual desires than I have had for some time. (Score if "No.") |
| 2 | I recently have had more trouble expressing my thoughts or saying what I want to say. |
| 1 | I feel like talking with people, even if it's necessary to phone them long distance. (Score if "No.") |
| 2 | I have been waking up earlier than I need to. |
| 3 | I am sleeping more than I used to. |
| 3 | Nowadays I feel worse in the morning. |
| 2 | My memory is not as sharp as it used to be. |
| 3 | My evenings and nights are my best times. |
| 2 | I am napping more than I used to. |
| 1 | Last night I had trouble getting to sleep. |
| 1 | Last night I woke up more than once. |
| | (Do you have any of the following symptoms?) |
| 1 | Drowsiness |
| 2 | Poor memory |
| 3 | Constipation |
| 3 | Decreased appetite |
| 3 | Lack of energy |

3. Organicity--Total 45 points

| Weight | Item |
|---|---|
| 2 | I simply don't have the energy to do things the way I used to. |
| 3 | When I meet people I feel I have nothing to say. |
| 2 | I find it increasingly difficult to make decisions. |
| 3 | I recently have had more trouble expressing my thoughts or saying what I want to say. |
| 2 | I am sleeping more than I used to. |
| 3 | My memory is not as sharp as it used to be. |
| 2 | My evenings and nights are my best times. (Score if "No.") |
| 1 | I am up all night and sleep during the day. |
| 3 | I am napping more than I used to. |
| | (Do you have any of the following symptoms?) |
| 3 | Drowsiness |
| 1 | Spasms of neck or tongue |
| 3 | Poor memory |
| 1 | Dizziness |
| 2 | Clumsiness |
| 1 | Fainting spells |
| 1 | Blurred vision |
| 1 | Headaches |
| 1 | Nausea or vomiting |
| 1 | Cough |
| 1 | Difficulty starting urination |
| 1 | Edema (swelling) of legs |
| 3 | Unsteady gait (poor balance) |
| 3 | Slurred speech |
| 1 | Chest pain |

4. Psychosis (P)--Total 29 points

| Weight | Item |
|---|---|
| 3 | If I watch very carefully, I can see or hear things other people can't. |
| 3 | Noises all seem to be louder to me than they were before. It's as if someone had turned up the volume. |
| 2 | I have a feeling that people are aginst me. |
| 3 | My thoughts are racing so fast that it frightens me. |
| 3 | I am having very strange experiences. |
| 3 | When I am talking to somebody, the slightest thing distracts me, even if a person only crosses his legs. |
| 1 | Sometimes I get frightened because everything seems far away. |
| 3 | When I am with certain people, very peculiar things begin to happen. |
| 3 | I suspect somebody is trying to make me do things. |
| 3 | At times I am not sure whether I am awake or dreaming. |
| 2 | I am up all night and sleep during the day. |

5. Neurosis (N)--Total 13 points

| Weight | Item |
|---|---|
| 3 | I have recently started to avoid riding trains, elevators, or bridges, or being in enclosed areas or high places. |
| 1 | I am having very strange experiences. |
| 3 | I cannot stop myself from checking things over and over. |
| 3 | Certain thoughts come in my mind and I can't keep them out. |
| 1 | Last night I slept soundly. (Score if "No.") |
| 2 | Last night I had trouble getting to sleep. |

6. Mania (M)--Total 26 points

| Weight | Item |
|---|---|
| 3 | I feel like going out and spending money. |
| 1 | I am enjoying my food. |
| 1 | I find that my sleeping has slowed down recently. (Score if "No.") |
| 1 | I find it increasingly difficult to make decisions. (Score if "No.") |
| 3 | I feel irritable wnen people interfere with my plans. |
| 3 | I feel that things are looking up and I can accomplish great things. |
| 2 | I now have more sexual desires than I have had for some time. |
| 1 | I recently have had more trouble expressing my thoughts or saying what I want to say. (Score if "No.") |
| 3 | I feel like talking to people, even if it's necessary to phone them long distance. |
| 2 | I have been waking up earlier than I need to. |
| 1 | I am sleeping more than I used to. (Score if "No.") |
| 3 | Lately I find myself losing my temper. |
| 2 | Last night I had difficulty getting to sleep. |

7. Impulsivity (I)--Total 14 points

| Weight | Item |
|---|---|
| 3 | I feel like going out and spending money. |
| 2 | I feel restless and cannot keep still. |
| 3 | I feel irritable when people interfere with my plans. |
| | (Do you have any of the following symptoms?) |
| 2 | Clumsiness |
| 3 | Increased sweating |
| 1 | Increased thirst |

8. Paranoia (PA)--Total 16 points

| Weight | Item |
|---|---|
| 3 | If I watch very carefully, I can see or hear things other people can't. |
| 1 | I am scared of going to new places or meeting new people. |
| 1 | I feel irritable when people interfere with my plans. |
| 3 | I have a feeling that people are against me. |
| 2 | I am having very strange experiences. |
| 3 | When I am with certain people, very peculiar things begin to happen. |
| 3 | I suspect someone is trying to make me do things. |

9. Suicidal Ideation (SU)--Total 32 points

| Weight | Item |
|---|---|
| 3 | I feel downhearted and blue. |
| 3 | When I meet people I feel I have nothing to say. |
| 1 | I feel restless and cannot keep still. |
| 1 | I am dreaming more now than I usually do. (Score if "No.") |
| 3 | I feel that others would be better off if I were dead. |
| 3 | I have been waking up earlier than I need to. |
| 2 | My memory is not as sharp as it used to be. |
| 1 | I am up all night and sleep during the day. |
| 2 | Last night I had difficulty getting to sleep. |
| | (Do you have any of the following symptoms?) |
| 2 | Poor memory |
| 1 | Headaches |
| 2 | Nervousness |
| 1 | Dry mouth and throat. |
| 3 | Nightmares |
| 3 | Decreased appetite |
| 1 | Increased sweating |

10. Chronic Anxiety (A3)--Total 13 points

| Weight | Item (Score if "Yes.") |
|---|---|
| 3 | Do your muscles tighten up when you become anxious? |
| 2 | Do you often get a headache when you become tense? |
| 1 | Do you often worry that something bad isgoing to happen? |
| 3 | Do you get anxious when you have to ask someone for a favor? |
| 1 | Are you concerned that people will think you are unattractive? |
| 3 | Do you feel bad for a long time when somebody criticizes you? |

11. Chronic Depression (D3)--Total 6 points

| Weight | Item (Score if "Yes.") |
|---|---|
| 1 | Have you often asked yourself, "Have I done the right thing?" |
| 1 | Are you concerned that people think you are unattractive? |
| 2 | Do you feel that your life is in a rut and that you are unable to change it? |
| 2 | Do you become tired and exhausted easily? |

12. Chronic Paranoia (PA3)--Total 7 points

| Weight | Item (Score if "Yes.") |
|---|---|
| 1 | Do you feel more relaxed indoors? |
| 1 | Do you stop and think before doing the smallest thing? |
| 1 | Do you feel that your life is in a rut and that you are unable to change it? |
| 3 | Do you find that deep down most people are full of hatred? |
| 1 | Do you get into fights with people you love? |

13. Chronic Impulsivity (I3)--Total 15 points

| Weight | Item (Score if "Yes.") |
|---|---|
| 3 | Do you like being in the midst of excitement? |
| 3 | Do you like making quick decisions? |
| 3 | Do you tend to lose your temper when you don't get your way? |
| 1 | Do you usually try to be the center of attention or the life of the party? |
| 1 | Are you often concerned that somehow you are going to lose control and hurt yourself or somebody else? |
| 3 | Do you tend to get into accidents? |
| 1 | Do you get bored with almost everything you do? |

14. Chronic Neuroticism (N3)--Total 15 points

| Weight | Item (Score if "Yes.") |
|---|---|
| 2 | Do you fear being left alone? |
| 3 | Do you feel panicky in crowds? |
| 2 | Do you often have periods during which everything and everybody seems unreal and far away? |
| 3 | Do you feel compelled to count things? |
| 2 | Do you often worry about your physical health? |
| 3 | Are you always afraid of catching somebody's illness or being contaminated? |

15. Chronic Psychopathy (PSY)--Total 9 points

| Weight | Item (Score if "Yes.") |
|---|---|
| 3 | Do you tend to be late for appointments? |
| 1 | Do you usually try to be the center of attention or the life of the party? |
| 1 | Do you often think about things that happened when you were a child? |
| 1 | Do you tend to get into accidents? |
| 3 | Do you get bored with almost everything you do? |

16. Chronic Suicidal Ideation (SUI)--Total 23 points

| Weight | Item (Score if "Yes" unless noted) |
|---|---|
| 2 | Do you like being in the midst of excitement? |
| 3 | Do you often feel like you are posing or pretending? |
| 1 | Do you fear being left alone? |
| 1 | Do you feel panicky in crowds? |
| 1 | Have you often asked yourself, "Have I done the right thing?" |
| 2 | Do you tend to lose your temper when you don't get your way? |
| 1 | Do you often get a heacache when you feel tense? |
| 1 | Do you often worry that something bad is going to happen? |
| 1 | Do you usually try to be the center of attention or the life of the party? |
| 2 | Are you concerned that people think you are unattractive? |
| 3 | Do you feel bad for a long time when somebody criticizes you? |
| 1 | Do you think people find you sexually attractive? (Score if "No.") |
| 1 | Do you get into fights with people you love? |
| 1 | Do you often think about things that happened when you were a child? |
| 2 | Do you tend to get into accidents? |

17. Personality Disorder (PER)--Total 23 points

| Weight | Item (Score if "Yes.") |
|---|---|
| 3 | Do you often feel you are posing or pretending? |
| 2 | Do you feel more relaxed indoors? |
| 1 | Do you fear being left alone? |
| 1 | Do you feel panicky in crowds? |
| 2 | Have you often asked yourself, "Have I done the right thing?" |
| 2 | Do you stop and think before doing the smallest thing? |
| 2 | Do you get anxious when you have to meet new people or go to a new place? |
| 2 | Do you become easily enthusiastic and just as easily disappointed about people? |
| 2 | Do you often feel annoyed with people who are not orderly? |
| 2 | Can you easily be cheered up even when you are very upset? |
| 1 | Do you often worry about your physical health? |
| 3 | Do you find it difficult to get close to people? |

# Appendix 3

## Distribution of the Scores

| Score | Anxiety | Depression | Organicity | Psychosis | Suicidal Ideation |
|---|---|---|---|---|---|
| 0 | 3 | 1 | 3 | 15 | 2 |
| 1 | 0 | 0 | 1 | 0 | 3 |
| 2 | 3 | 3 | 2 | 4 | 2 |
| 3 | 1 | 0 | 2 | 12 | 5 |
| 4 | 4 | 0 | 0 | 3 | 3 |
| 5 | 4 | 1 | 1 | 5 | 1 |
| 6 | 1 | 3 | 1 | 5 | 3 |
| 7 | 2 | 1 | 3 | 0 | 3 |
| 8 | 3 | 0 | 1 | 3 | 2 |
| 9 | 3 | 1 | 1 | 3 | 3 |
| 10 | 2 | 1 | 1 | 0 | 4 |
| 11 | 2 | 1 | 3 | 3 | 4 |
| 12 | 1 | 4 | 2 | 0 | 3 |
| 13 | 2 | 3 | 2 | 1 | 4 |
| 14 | 4 | 2 | 3 | 2 | 4 |
| 15 | 5 | 3 | 2 | 0 | 4 |
| 16 | 2 | 0 | 1 | 0 | 2 |
| 17 | 3 | 4 | 5 | 2 | 1 |
| 18 | 0 | 3 | 3 | 0 | 3 |
| 19 | 3 | 2 | 5 | 1 | 1 |
| 20 | 3 | 4 | 1 | 0 | 1 |
| 21 | 3 | 0 | 1 | 2 | 3 |
| 22 | 0 | 2 | 3 | 1 | 0 |
| 23 | 1 | 0 | 3 | 0 | 0 |
| 24 | 0 | 0 | 0 | 0 | 0 |
| 25 | 1 | 1 | 2 | 0 | 1 |
| 26 | 1 | 1 | 0 | 0 | 1 |
| 27 | 2 | 3 | 3 | 1 | 1 |
| 28 | 1 | 2 | 1 | 0 | |
| 29 | 1 | 2 | 2 | 1 | |
| 30 | 1 | 2 | 4 | | |
| 31 | 1 | 3 | 0 | | |
| 32 | 0 | 2 | 1 | | |
| 33 | 1 | 2 | 0 | | |
| 34 | | 2 | 0 | | |
| 35 | | 3 | 0 | | |
| 36 | | 0 | 1 | | |
| 37 | | 0 | | | |
| 38 | | 1 | | | |
| 39 | | 0 | | | |
| 40 | | 0 | | | |
| 41 | | 0 | | | |
| 42 | | 0 | | | |
| 43 | | 1 | | | |

| Score | Neurosis | Mania | Impulsivity | Paranoia | Chronic Anxiety | Chronic Depression |
|---|---|---|---|---|---|---|
| 0 | 15 | 0 | 8 | 13 | 4 | 7 |
| 1 | 1 | 1 | 1 | 13 | 1 | 7 |
| 2 | 3 | 0 | 4 | 3 | 4 | 7 |
| 3 | 15 | 3 | 8 | 4 | 7 | 11 |
| 4 | 2 | 7 | 4 | 11 | 4 | 7 |
| 5 | 3 | 2 | 8 | 3 | 8 | 12 |
| 6 | 12 | 3 | 7 | 4 | 4 | 13 |
| 7 | 3 | 6 | 3 | 1 | 7 | |
| 8 | 8 | 3 | 6 | 4 | 3 | |
| 9 | 2 | 5 | 7 | 0 | 9 | |
| 10 | 0 | 10 | 3 | 2 | 7 | |
| 11 | 2 | 4 | 4 | 0 | 0 | |
| 12 | 4 | 1 | 0 | 1 | 2 | |
| 13 | | 4 | 0 | 1 | 4 | |
| 14 | | 5 | 1 | 0 | | |
| 15 | | 4 | | 2 | | |
| 16 | | 3 | | 2 | | |
| 17 | | 0 | | | | |
| 18 | | 2 | | | | |
| 19 | | 0 | | | | |
| 20 | | 1 | | | | |

| Score | Chronic Impuls. | Chronic Neurotic. | Chronic Paranoia | Personality Disorder | Chronic Psychopathy | Chronic Sui. Id. |
|---|---|---|---|---|---|---|
| 0 | 7 | 10 | 4 | 1 | 15 | 2 |
| 1 | 4 | 0 | 11 | 0 | 17 | 1 |
| 2 | 1 | 14 | 17 | 0 | 8 | 0 |
| 3 | 11 | 4 | 12 | 2 | 3 | 5 |
| 4 | 8 | 0 | 7 | 2 | 8 | 6 |
| 5 | 8 | 13 | 7 | 2 | 7 | 4 |
| 6 | 7 | 2 | 5 | 5 | 3 | 3 |
| 7 | 4 | 9 | 1 | 2 | 3 | 4 |
| 8 | 5 | 3 | | 5 | | 6 |
| 9 | 2 | 0 | | 6 | | 5 |
| 10 | 3 | 4 | | 13 | | 7 |
| 11 | 0 | 0 | | 5 | | 6 |
| 12 | 2 | 4 | | 2 | | 2 |
| 13 | 1 | 0 | | 4 | | 5 |
| 14 | 0 | 0 | | 6 | | 5 |
| 15 | 1 | 1 | | 3 | | 0 |
| 16 | | | | 2 | | 0 |
| 17 | | | | 0 | | 0 |
| 18 | | | | 4 | | 1 |
| 19 | | | | | | 0 |
| 20 | | | | | | 1 |
| 21 | | | | | | 1 |

# Appendix 4

## Correlations Between Measured Parameters

The correlation coefficients between data of linear variation are presented as Appendix 4. In this table the abbreviations used are as follows:

| Abbreviation | Meaning |
|---|---|
| Socio | Socioeconomic class |
| Onset | Type of Onset |
| Age | Age of Onset |
| Duration | Duration of epilepsy |
| Hist Control | History of seizure control |
| Freq | Seizure frequency |
| Psych1 | History of psychotherapy |
| Psych2 | History of psychiatric hosp. |
| Dilantin | Diphenylhydantoin dosage |
| Phenobar | Phenobarbital dosage |
| Mysoline | Primidone dosage |

The abbreviations A, D, P, O, N, M, I, PA, SU, A3, D3, I3, N3, PA3, PER, PSY, and SUI are defined in Materials and Methods and Appendix 2. These entities represent the scores on scales of symptoms which are given as Appendix 2.

## CORRELATION COEFFICIENTS

| VARIABLE DESCRIPTION | NAME | VAR (1) | VAR (2) | VAR (3) | VAR (4) | VAR (5) | VAR (6) | VAR (7) |
|---|---|---|---|---|---|---|---|---|
| SOCIO | VAR(1) | 1.000 | -0.154 | -0.123 | -0.070 | -0.134 | 0.155 | 0.022 |
| ONSET | VAR(2) | -0.154 | 1.000 | -0.245 | 0.018 | -0.037 | -0.055 | -0.067 |
| AGE | VAR(3) | -0.123 | -0.245 | 1.000 | -0.408 | 0.113 | 0.095 | 0.165 |
| DURATION | VAR(4) | -0.070 | 0.018 | -0.408 | 1.000 | -0.035 | 0.155 | 0.048 |
| HIST CONTRL | VAR(5) | -0.134 | -0.037 | 0.113 | -0.035 | 1.000 | -0.537 | -0.126 |
| FREQ | VAR(6) | 0.155 | -0.055 | 0.095 | 0.155 | -0.537 | 1.000 | 0.221 |
| PSYCH1 | VAR(7) | 0.022 | -0.067 | 0.165 | 0.048 | -0.126 | 0.221 | 1.000 |
| PSYCH2 | VAR(8) | -0.231 | 0.124 | -0.198 | 0.169 | 0.056 | -0.059 | -0.440 |
| DILANTIN | VAR(9) | -0.044 | 0.160 | 0.026 | 0.115 | -0.123 | 0.217 | 0.043 |
| PHENOBAR | VAR(10) | -0.043 | -0.040 | -0.014 | 0.151 | -0.125 | 0.082 | 0.058 |
| MYSOLINE | VAR(11) | 0.199 | 0.203 | -0.267 | 0.082 | 0.259 | -0.297 | -0.154 |
| A | VAR(12) | -0.250 | -0.035 | 0.117 | -0.029 | 0.019 | -0.246 | -0.089 |
| D | VAR(13) | -0.243 | -0.066 | 0.152 | -0.028 | 0.120 | -0.316 | -0.173 |
| P | VAR(14) | -0.298 | -0.057 | -0.026 | -0.111 | -0.072 | -0.126 | -0.211 |
| O | VAR(15) | -0.196 | -0.135 | 0.177 | -0.130 | 0.160 | -0.329 | -0.126 |
| N | VAR(16) | -0.296 | -0.009 | -0.006 | 0.001 | 0.040 | -0.167 | -0.271 |
| M | VAR(17) | -0.052 | -0.057 | -0.123 | -0.152 | 0.049 | -0.284 | -0.216 |
| I | VAR(18) | -0.069 | -0.074 | -0.024 | -0.101 | 0.082 | -0.268 | -0.213 |
| PA | VAR(19) | -0.185 | 0.010 | -0.130 | -0.025 | -0.142 | -0.078 | -0.201 |
| SU | VAR(20) | -0.319 | 0.013 | 0.013 | -0.019 | 0.083 | -0.341 | -0.173 |
| A3 | VAR(21) | -0.150 | 0.023 | 0.163 | -0.074 | 0.028 | -0.263 | -0.172 |
| D3 | VAR(22) | -0.180 | -0.161 | 0.124 | -0.033 | 0.129 | -0.171 | -0.202 |
| I3 | VAR(23) | 0.031 | -0.052 | 0.070 | -0.282 | 0.188 | -0.196 | -0.051 |
| N3 | VAR(24) | -0.150 | -0.173 | 0.079 | -0.147 | -0.002 | -0.194 | -0.136 |
| PA3 | VAR(25) | -0.255 | 0.040 | -0.033 | -0.051 | 0.019 | -0.212 | -0.202 |
| PER | VAR(26) | -0.156 | 0.217 | 0.026 | 0.084 | -0.032 | -0.158 | -0.205 |
| PSY | VAR(27) | -0.128 | -0.055 | 0.061 | -0.175 | 0.147 | -0.193 | -0.090 |
| SUI | VAR(28) | -0.158 | -0.006 | 0.207 | -0.269 | 0.169 | -0.385 | -0.199 |

CORRELATION COEFFICIENTS

| VARIABLE DESCRIPTION | NAME | VAR (8) | VAR (9) | VAR (10) | VAR (11) | VAR (12) | VAR (13) | VAR (14) |
|---|---|---|---|---|---|---|---|---|
| SOCIO | VAR(1) | -0.231 | -0.044 | -0.043 | 0.199 | -0.250 | -0.243 | -0.298 |
| ONSET | VAR(2) | 0.124 | 0.160 | -0.040 | 0.203 | -0.085 | -0.066 | -0.057 |
| AGE | VAR(3) | -0.198 | 0.026 | -0.014 | -0.267 | 0.117 | 0.152 | -0.026 |
| DURATION | VAR(4) | 0.169 | 0.115 | 0.151 | 0.082 | -0.029 | -0.028 | -0.111 |
| HIST CONTRL | VAR(5) | 0.056 | -0.123 | -0.125 | 0.259 | 0.019 | 0.120 | -0.072 |
| FREQ | VAR(6) | -0.059 | 0.217 | 0.082 | -0.297 | -0.246 | -0.316 | -0.126 |
| PSYCH1 | VAR(7) | -0.440 | 0.043 | 0.058 | -0.154 | -0.089 | -0.173 | -0.211 |
| PSYCH2 | VAR(8) | 1.000 | -0.207 | 0.052 | 0.019 | 0.015 | 0.031 | 0.217 |
| DILANTIN | VAR(9) | -0.207 | 1.000 | 0.047 | 0.027 | 0.083 | 0.006 | -0.210 |
| PHENOBAR | VAR(10) | 0.052 | 0.047 | 1.000 | -0.635 | 0.056 | 0.111 | -0.047 |
| MYSOLINE | VAR(11) | 0.019 | 0.027 | -0.635 | 1.000 | -0.200 | -0.112 | -0.165 |
| A | VAR(12) | 0.015 | 0.083 | 0.056 | -0.200 | 1.000 | 0.636 | 0.572 |
| D | VAR(13) | 0.031 | 0.006 | 0.111 | -0.112 | 0.636 | 1.000 | 0.622 |
| P | VAR(14) | 0.217 | -0.210 | -0.047 | -0.165 | 0.572 | 0.622 | 1.000 |
| O | VAR(15) | -0.007 | -0.029 | 0.098 | -0.190 | 0.720 | 0.871 | 0.573 |
| N | VAR(16) | 0.212 | -0.066 | -0.046 | -0.088 | 0.522 | 0.530 | 0.715 |
| M | VAR(17) | 0.198 | -0.018 | -0.132 | 0.070 | 0.376 | 0.346 | 0.412 |
| I | VAR(18) | 0.102 | 0.013 | -0.097 | 0.005 | 0.539 | 0.570 | 0.548 |
| PA | VAR(19) | 0.230 | -0.193 | -0.105 | -0.034 | 0.359 | 0.492 | 0.675 |
| SU | VAR(20) | 0.007 | 0.056 | 0.134 | -0.157 | 0.748 | 0.859 | 0.608 |
| A3 | VAR(21) | 0.108 | -0.201 | 0.142 | -0.112 | 0.307 | 0.306 | 0.222 |
| D3 | VAR(22) | -0.035 | 0.121 | -0.008 | -0.069 | 0.487 | 0.478 | 0.371 |
| I3 | VAR(23) | -0.090 | -0.209 | -0.057 | 0.059 | 0.106 | 0.123 | 0.171 |
| N3 | VAR(24) | -0.125 | -0.181 | 0.038 | -0.140 | 0.259 | 0.402 | 0.346 |
| PA3 | VAR(25) | 0.028 | -0.127 | 0.098 | -0.035 | 0.267 | 0.346 | 0.357 |
| PER | VAR(26) | -0.005 | 0.129 | 0.106 | 0.018 | 0.154 | 0.279 | 0.071 |
| PSY | VAR(27) | -0.158 | -0.016 | -0.066 | -0.010 | 0.382 | 0.303 | 0.252 |
| SUI | VAR(28) | -0.087 | -0.132 | -0.005 | 0.030 | 0.331 | 0.465 | 0.324 |

| VARIABLE DESCRIPTION | NAME | VAR (15) | VAR (16) | VAR (17) | VAR (18) | VAR (19) | VAR (20) | VAR (21) |
|---|---|---|---|---|---|---|---|---|
| SOCIO | VAR(1) | -0.196 | -0.296 | -0.052 | -0.069 | -0.185 | -0.319 | -0.150 |
| ONSET | VAR(2) | -0.135 | -0.009 | -0.057 | -0.074 | 0.010 | 0.013 | 0.023 |
| AGE | VAR(3) | 0.177 | -0.006 | -0.123 | -0.024 | -0.130 | 0.013 | 0.163 |
| DURATION | VAR(4) | -0.130 | 0.001 | -0.152 | -0.101 | -0.025 | -0.019 | -0.074 |
| HIST CONTRL | VAR(5) | 0.180 | 0.040 | 0.049 | 0.082 | -0.142 | 0.083 | 0.028 |
| FREQ | VAR(6) | -0.329 | -0.167 | -0.284 | -0.268 | -0.078 | -0.341 | -0.263 |
| PSYCH1 | VAR(7) | -0.126 | -0.271 | -0.216 | -0.213 | -0.201 | -0.173 | -0.172 |
| PSYCH2 | VAR(8) | -0.007 | 0.212 | 0.198 | 0.102 | 0.230 | 0.007 | 0.108 |
| DILANTIN | VAR(9) | -0.029 | -0.068 | -0.018 | 0.013 | -0.198 | 0.066 | -0.201 |
| PHENOBAR | VAR(10) | 0.098 | -0.046 | -0.132 | -0.097 | -0.105 | 0.134 | 0.142 |
| MYSOLINE | VAR(11) | -0.130 | -0.088 | 0.070 | 0.005 | -0.034 | -0.157 | -0.112 |
| A | VAR(12) | 0.720 | -0.522 | -0.376 | 0.539 | -0.359 | 0.748 | -0.307 |
| D | VAR(13) | 0.871 | -0.530 | -0.346 | 0.570 | -0.492 | -0.859 | -0.306 |
| P | VAR(14) | 0.573 | -0.715 | -0.412 | -0.548 | -0.375 | -0.608 | -0.222 |
| O | VAR(15) | 1.000 | -0.503 | -0.423 | -0.596 | -0.424 | -0.821 | -0.362 |
| N | VAR(16) | -0.503 | 1.000 | -0.468 | -0.522 | -0.556 | -0.608 | -0.145 |
| M | VAR(17) | -0.423 | -0.468 | 1.000 | -0.754 | -0.391 | -0.406 | -0.190 |
| I | VAR(18) | -0.596 | -0.522 | -0.754 | 1.000 | -0.455 | -0.563 | -0.271 |
| PA | VAR(19) | -0.424 | -0.566 | -0.391 | -0.455 | 1.000 | -0.457 | -0.249 |
| SU | VAR(20) | -0.821 | -0.608 | -0.406 | -0.563 | -0.457 | 1.000 | -0.347 |
| A3 | VAR(21) | -0.362 | -0.145 | -0.190 | -0.271 | -0.249 | -0.347 | 1.000 |
| D3 | VAR(22) | -0.516 | -0.344 | -0.388 | -0.433 | -0.325 | -0.489 | -0.489 |
| I3 | VAR(23) | -0.198 | -0.093 | -0.332 | -0.191 | -0.168 | -0.133 | -0.315 |
| N3 | VAR(24) | -0.439 | -0.285 | -0.223 | -0.307 | -0.307 | -0.433 | -0.524 |
| PA3 | VAR(25) | -0.218 | -0.252 | -0.164 | -0.193 | -0.341 | -0.372 | -0.418 |
| PER | VAR(26) | -0.216 | -0.047 | -0.156 | -0.206 | -0.156 | -0.345 | -0.535 |
| PSY | VAR(27) | -0.426 | -0.209 | -0.209 | -0.273 | -0.163 | -0.378 | -0.321 |
| SUI | VAR(28) | -0.486 | -0.248 | -0.366 | -0.370 | -0.359 | -0.448 | -0.710 |

## CORRELATION COEFFICIENTS

| VARIABLE DESCRIPTION | NAME | VAR (22) | VAR (23) | VAR (24) | VAR (25) | VAR (26) | VAR (27) | VAR (28) |
|---|---|---|---|---|---|---|---|---|
| SOCIO | VAR(1) | -0.180 | 0.031 | -0.150 | -0.255 | -0.156 | -0.128 | -0.158 |
| ONSET | VAR(2) | -0.161 | -0.032 | -0.173 | 0.040 | 0.217 | -0.055 | -0.006 |
| AGE | VAR(3) | 0.124 | 0.070 | 0.079 | -0.033 | 0.026 | 0.061 | 0.207 |
| DURATION | VAR(4) | -0.033 | -0.282 | -0.147 | -0.051 | 0.084 | -0.175 | -0.259 |
| HIST CONTRL | VAR(5) | 0.129 | 0.188 | -0.002 | 0.019 | -0.032 | 0.147 | 0.169 |
| FREQ | VAR(6) | -0.171 | -0.196 | -0.194 | -0.212 | -0.158 | -0.193 | -0.385 |
| PSYCH1 | VAR(7) | -0.202 | -0.051 | -0.136 | -0.202 | -0.205 | -0.090 | -0.199 |
| PSYCH2 | VAR(8) | -0.035 | -0.090 | -0.125 | 0.028 | -0.005 | -0.158 | -0.087 |
| DILANTIN | VAR(9) | 0.121 | -0.209 | -0.181 | -0.127 | 0.129 | -0.016 | -0.132 |
| PHENOBAR | VAR(10) | -0.008 | -0.057 | 0.038 | 0.093 | 0.105 | -0.066 | -0.005 |
| MYSOLINE | VAR(11) | -0.069 | 0.059 | -0.140 | -0.035 | 0.018 | -0.010 | 0.030 |
| A | VAR(12) | 0.487 - | 0.106 | 0.259 | 0.267 | 0.154 | 0.382 | 0.331 |
| D | VAR(13) | 0.478 - | 0.123 | 0.402 - | 0.346 | 0.279 | 0.303 | 0.405 |
| P | VAR(14) | 0.371 | 0.171 | 0.346 | 0.357 | 0.071 | 0.252 | 0.324 |
| O | VAR(15) | 0.516 - | 0.198 | 0.439 - | 0.218 | 0.216 | 0.426 - | 0.486 |
| N | VAR(16) | 0.344 | 0.093 | 0.285 | 0.252 | 0.047 | 0.209 | 0.248 |
| M | VAR(17) | 0.388 | 0.332 | 0.223 | 0.164 | 0.156 | 0.209 | 0.366 |
| I | VAR(18) | 0.433 - | 0.171 | 0.307 | 0.173 | 0.206 | 0.273 | 0.370 |
| PA | VAR(19) | 0.325 | 0.158 | 0.307 | 0.341 | 0.156 | 0.163 | 0.359 |
| SU | VAR(20) | 0.453 - | 0.133 | 0.433 - | 0.372 | 0.345 | 0.378 | 0.448 |
| A3 | VAR(21) | 0.439 - | 0.315 | 0.524 - | 0.418 - | 0.555 - | 0.321 | 0.710 |
| D3 | VAR(22) | 1.000 | 0.318 | 0.454 - | 0.567 - | 0.472 - | 0.442 - | 0.604 |
| I3 | VAR(23) | 0.318 | 1.000 | 0.292 | 0.227 | 0.222 | 0.482 - | 0.680 |
| N3 | VAR(24) | 0.454 - | 0.292 | 1.000 | 0.553 - | 0.540 - | 0.404 - | 0.616 |
| PA3 | VAR(25) | 0.567 - | 0.227 | 0.553 - | 1.000 | 0.630 - | 0.362 | 0.540 |
| PER | VAR(26) | 0.472 - | 0.222 | 0.540 - | 0.630 - | 1.000 | 0.335 | 0.590 |
| PSY | VAR(27) | 0.442 - | 0.482 - | 0.404 - | 0.362 | 0.335 | 1.000 | 0.551 |
| SUI | VAR(28) | 0.604 - | 0.680 - | 0.616 - | 0.540 - | 0.590 - | 0.551 - | 1.000 |

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